

Issues in Environmental Health:  
The Case of pesticides.

**Community Health Cell**  
*Library and Documentation Unit*  
**BANGALORE**

8761

COMMUNITY HEALTH CELL  
326, V Main, I Block  
Koramangala  
Bangalore-560034  
India



# ISSUES IN ENVIRONMENTAL HEALTH: THE CASE OF PESTICIDES

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### INTRODUCTION

It is now more than a year since the Bhopal disaster occurred and we are left with no words with which to express our anger, our concern and our condemnation of the callousnees of the industry and the authorities. For many groups like the Medico Friend Circle the past year has also meant a reassessment of strengths and weaknesses against the background of Bhopal. We have acutely felt the lack of access to vital, life-saving information and have regretted the inability of the voluntary sector to unearth and circulate relevant information in areas such as environmental health.

As a response to this realisation the XII Annual Meet of the Medico Friend Circle will focus on "Issues in Environmental Health : A Case Study of Pesticides". We hope that this broader forum of the annual meet will help us to understand many of the issues which we as individuals and groups have had to confront in recent times. This set of papers is meant to serve as a general backgrounder for the conference. Some of these contributions put together a gamut of useful information, some raise critical issues, while others provide perspective views.

The compilation begins with a general overview of environmental health in India followed by a set of papers specifically focussing on pesticides and their impact on human health. A second set deals with the problems involved in evaluating and deriving relevant information from existing studies and reports. The politics of pesticides and a review of existing legislation have also been discussed in two separate papers.

This compilation is being produced in collaboration with the Centre for Education and Documentation, Bombay. We hope the issues raised will be discussed at the Meet and outside it. If sufficient response is received a future edition will be brought out which will incorporate the discussions that are generated by these papers at the XII Annual Meet of the Medico Friend Circle.

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"India faces a disastrous 'double burden' of disease. Most old diseases continue to be rampant while new ones are making rapid strides."

- *State of India's Environment*  
A Citizens' Report, 1982

Unceasing environmental degradation has had disastrous consequences on the health of the people of India. Bhopal was only a moment, albeit an eye opening one, in the long history of disgrace heaped upon the environment as a result of explosive and blind industrialisation for high profits, and of unplanned urbanisation as its inevitable outcome. Murky rivers, foul air, congested housing, jammed roads, deadly workplaces -- in industries, in agriculture and at home, disease producing agricultural development, denuded forests alongwith poverty, inequality, ignorance and oppression have taken a heavy toll of the physical, psychological and social health of Indian people.

The peculiar nature of development in India has produced a paradoxical situation. One, there is a co-existence of the diseases of antiquity -- infectious diseases with those of modernity -- pollution and stress related diseases. This phenomenon is seen at its worst in urban areas. In cities, the effects of faecal pollution of drinking water is compounded by pollution by industrial effluents - air borne infection as well as chemical pollutants abound in the atmosphere. The situation in the rural areas too, is fast deteriorating. Traditional water sources are drying out as a result of extensive deforestation and silting, while modern irrigation has brought in newer mosquito borne diseases and problems like knock knee syndrome.

The problem is a vast and a complex one. It is intrinsically woven into the whole process of social development, and therefore, cannot have patch-work solutions. An attempt is made here to present an overall picture of human health and environment in India. The categorisation resorted to is an arbitrary one and is done so for descriptive ease. In reality, these categories exist in an interwoven, inter-reacting fashion.

## I. Water

### Bacterial Pollution

According to scientists at National Environmental Engineering Research Institute (NEERI), about 70 per cent of available water in India is polluted. Most of the human wastes are discharged untreated into water sources. Out of India's 3119 towns and cities, only 217 have full or partial sewerage and sewage treatment facilities. Since sewers and water mains run side by side, raw sewage through leaks can get sucked into the water mains when the pressure is low. According to the chief water analyst of Tamil Nadu, reporting in Madras, 96.5 per cent of samples tested showed recent faecal contamination.

In rural areas, the situation is even worse. In 1971-72, 60,000 villages out of a total of 5,76,000 were identified as health problem villages - with problems endemic to water borne infectious diseases or those where available water has an excess of salinity, iron or toxic elements. Before the Yamuna enters Delhi, its water contains 7,500 MPN/100 ml. of coliform bacteria, but further downstream at Okhla it contains 24,00,000 MPN/100 ml., according to a study done by the Central Board for Prevention and Control of Water Pollution. The Indian Standard is under 5,000 MPN/100 ml. Most of the acute diseases affecting Indians are water borne - diarrhoeas, amoebic dysentery, cholera, typhoid and infective hepatitis (jaundice). It is estimated that 73 million work days are lost every year due to water-related diseases.

### Industrial Pollution

A series of investigations by the Institute of Science in Bombay shows that industries on the banks of the Kalu river on the outskirts of Bombay are recreating the Minamata disaster. The effluents are from industries which include a rayon factory, a paper mill, a dye factory and a chemical plant. The study shows that the water at the point of discharge has a very high content of mercury. The mercury salts in the effluent, which are insoluble,

## ENVIRONMENTAL POLLUTION AND HUMAN HEALTH An Overview Of The Indian Situation

Dhruv Mankad

Table 1 - Pollution Control Standards

Characteristics	Sewage IS4764-73	Industrial Effluents			Inland surface water OS 2296-74
		Inland surface water IS2490-74	Public sewers IS3306-74		
BOD (5 day 20°C) mg/l	20	30.0	500.00	3.0	-
COD mg/l	-	250.0	-	-	-
pH	-	5.5-9.0	5.5-9.0	6.9-9.0	-
Total suspended solids, mg/l	30	100.0	600.0	-	-
Temperature, °C	-	40.0	45.0	-	-
Oil & grease, mg/l	-	10.0	100.0	0.1	-
Phenolic compounds, mg/l	-	1.0	5.0	0.005	-
Cyanides, mg/l	-	0.2	2.0	0.01	-
Sulphides, mg/l	-	2.0	-	1.5	-
Fluorides, mg/l	-	2.0	-	-	-
Total residual chlorine, mg/l	-	1.0	-	-	-
Insecticides, mg/l	-	Zero	-	Zero	-
Arsenic, mg/l	-	0.2	-	0.2	-
Zinc, mg/l	-	5.0	15.0	-	-
Chloride, mg/l	-	-	600.0	600.0	-
Sulphates, mg/l	-	-	-	1000.0	-
Ammonical nitrogen, mg/l	-	50.0	50.0	-	-
Nitrates, mg/l	-	-	-	50.0	-
Radioactive materials :					
Alpha-emitters, uc/ml	-	10 <sup>-7</sup>	-	10 <sup>-9</sup>	-
Beta-emitters, uc/ml	-	10 <sup>-6</sup>	-	10 <sup>-8</sup>	-
Dissolved oxygen, mg/l	-	-	-	40% of saturation value or 3 mg/l whichever is higher	-
Coliform organisms (monthly average) - MPN per 100 ml.	-	-	-	-	Under 5000

Source: Deodhar and Nath, "Water pollution and its control in India" Indian Science Congress 1981.

Table 2 - Water Pollution from Selected Industries

INDUSTRY	Waste Water generated (million m <sup>3</sup> / year)	POLLUTANTS				REMARKS
		BOD (mg/l)	COD (mg/l)	Suspended solids (mg/l)	Others (mg/l)	
Cotton textile	1,530	200-300	400-800	350-550	Chlorides 500-1,400	Highly alkaline, discoloured and hot effluent
Nitrogenous fertiliser	52	-	-	-	Total nitrogen 1,250 Fluoride 15 Arsenic 1.6 Phosphates 70	Nitrogen in ammonia is deadly to fish, nitrates are hazardous to livestock and humans, effluents are highly alkaline and slightly brownish.
Paper	450	160	725	410	-	High or low pH depending on process used.
Pesticides	-	Upto 700	3,000- 10,000	-	Organic nitrate 0-500 Sulphates 3,000-20,000	pH can vary from acid (2) to alkaline (10) effluent is highly toxic to aquatic life.
Oil refinery	15	160	320	350	Phenol 20 Sulphides 9 Phosphates 49	-
Integrated steel plants	40	75-100	200-2,000	-	Phenol 20-60	Acidic effluent high in suspended solids.
Sugar	225	300-2,000	600-4,000	220-800	Total nitrogen 10-40	-

Source : Deodhar and Nath "Water pollution and its control in India", Indian Science Congress, 1981.

Table 4 - Selected Atmospheric Pollutants

POLLUTANT	PRINCIPAL HUMAN SOURCES	EFFECTS	REMARKS
CARBON DIOXIDE	Fuel combustion for heating, transport, energy production.	No direct effect on people. Over time, may lead to increase in earth's temperature.	Normal constituent of atmosphere. Essential to plant life.
CARBON MONOXIDE	Incomplete fuel combustion (as in motor vehicles).	Deprives tissues of oxygen. People with cardio-respiratory diseases more sensitive.	Contribution of natural sources small. Smoking more significant for humans than exposure to traffic.
SULPHUR	Burning of sulphur-containing fuels like coal and oil.	Combined with smoke increases risk and effects of respiratory diseases. Causes suffocation, irritation of throat and eyes. Combines with atmospheric water vapour to produce acid rain. Reduces crop yields. Leads to acidification of lakes and soils. Corrodes buildings.	
SUSPENDED PARTICULATE MATTER	Smoke from domestic industrial and vehicular sources.	Possible toxic effects depend on specific composition. Aggravates effects of sulphur dioxide. Reduces sunlight and visibility. Increases corrosion.	Chemically, a most diverse group of substances. Natural sources include dust-storms, volcanic eruptions & sea spray.
OXIDES OF NITROGEN	Fuel combustion in motor vehicles and furnaces, forest fires.	Possible increase in acute respiratory infections and bronchitis morbidity in children. Produces brown haze in city air. Causes corrosion.	Nitrogen oxide and nitrogen dioxide are the two components.
VOLATILE HYDRO CARBONS	Partial combustion of carbonaceous fuels, industrial processes, disposal of solid wastes.	React with other pollutants to produce eye irritants (acrolein, aldehydes). Ethylene is harmful to plants. Aerosol particles reduce visibility. May produce unpleasant odours.	
OXIDANTS AND OZONE	Emissions from motor vehicles. Photochemical reactions of nitrogen oxides and reactive hydrocarbons.	Cause eye irritation and impaired pulmonary function in diseased persons. Corrode materials and reduce visibility. Ozone is one of the most damaging pollutants for plants.	Mainly derivative: products of atmospheric reactions between other pollutants. Ozone is a natural and essential constituent of the upper atmosphere.

Source : Preparatory document 48/8, Conference on the Human Environment, Stockholm 1972.

are converted to soluble mercurial compounds by certain bacteria. These in turn enter the food chain. The fish get poisoned and milch cattle that graze on plants in shallow water nearby accumulate mercury. It is found that there are 5 ppm of mercury in milk of the cattle feeding on the Pycreus plant growing in the river. A child drinking one litre of this milk every day consumes 35 ppm of mercury per week. Over a period of several months this can lead to accumulation of more than 0.3 mg of mercury, which is well above the safe level. It is reported that symptoms and signs of chronic mercury poisoning are already discernible in the people here.

In another case, the effluents from Harihar Polyfibres in Dharwad district of Karnataka are dumped into the Tungabhadra which has turned brownish red and full of froth. The bubbles on the surface keep bursting and the alkaline spray causes skin eruptions and conjunctivitis. Villagers who use water downstream complain of frequent headaches and back pains. They reportedly suffer from an unknown intestinal malady requiring costly surgical treatment.

Another source of water pollution is water from mines. For example, the wastes in Bastar, U.P. and from the Bailadilla iron ore mines pollute the Sarkhir river and affect nearby villages with a total population of 4,00,000. Continuous dewatering of the mines leads to flooding, waterlogging, silting and pollution. It also lowers the surrounding water table. For instance, the underground coal mines at Birshingpur of Shahdol district, M.P. pump out nearly 3,000 litres of water into a nullah. As a result the water table in the surrounding area dropped, resulting in crop failures. Reports indicate that if the water pumped out is supplied to townships, as done from the coal mines near Chhindwara, it can cause diseases relating to calcination.

## II. Atmosphere

Activities like industrial production, motor transport and domestic burning of fuel are adding harmful substances to the atmosphere.

Table 3 - Basic data on air quality in selected cities  
(in micrograms per cubic metre)

City	Suspended particulate		Sulphur di-oxide		Oxides of Nitrogen	
	1970	1979	1970	1979	1970	1979
Bombay	241	275	47	83	NA	23
Calcutta	341	578	33	85	NA	24
Delhi	601	481	41	39	NA	38
Madras	101	106	8	16	NA	12
Max. acceptable level (ISI)	150		60	Not yet established		

Sources : For 1970 Data CPHERI, Nagpur. For 1978-79 Data, NEERI. Air quality in selected cities of India, Nagpur.

The major pollutants are sulphur dioxide, oxides of nitrogen, suspended particulate matter and metallic traces. Industries using fossil fuels, oil and coal, thermal power stations, fertiliser plants, textile mills and land traffic are some of the major sources of air pollution. The common sources and their ill effects on human health are listed in Table 4.

Other pollutants like cotton dust from textile mills create hazards for the workers inside the factory (see Section on Workplace) as well as for people residing nearby. Continuous inhalation causes byssiosis, weakened respiratory function and lowered resistance to disease like TB and chronic bronchitis.

Air pollution generated by blasting machinery in mines leads to respiratory diseases and eye ailments. For instance, according to a survey conducted by the ILO and the Assistant Director General of Mines in November 1978 not only the lives of the mine workers but also of those living in surrounding areas. Clustering of mine related industries like ore concentrators, crushers and smelters also cause air pollution. The entire stretch between Faridabad and Badarpur outside Delhi is covered with a

thick layer of silica dust from stone quarries in the area. Visibility is reduced and breathing is difficult.

## Traffic Pollution

There are 3.7 million motor vehicles on the roads according to the National Transport Policy document (1980). Their exhaust fumes contain carbon monoxide, nitrogen oxides, hydrocarbons, aldehydes and lead oxides. The black smoke of diesel engines contains more particulates than the exhaust of petrol engines, but carbon monoxide hydrocarbons and aldehydes are less. According to Prof. J.N. Dave, Dean, School of Environmental Sciences, J.N.U. New Delhi, automobile emissions in Bombay and Delhi account for 70 per cent of the carbon monoxide, 50 per cent of the hydrocarbons and 30-40% of the particulates in the atmosphere. Carbon monoxide reduces the oxygen-carrying capacity of blood. Constant or prolonged inhalation of the gas poses serious threat to people with cardiovascular disorders. Lead is another toxic element present in automobile exhaust. A survey by NIOH, Ahmedabad indicated that the lead concentration in the blood and urine of policemen and roadside shopkeepers was significantly higher than that of unexposed people.

The terrible effects of traffic on the air are evident from a detailed study of 4,000 people conducted by Dr.S.R. Kamath of KEM Hospital and Air Pollution Prevention Cell of Bombay Municipal Corporation in 1977-78. The health of the residents of Khar -- a clear suburb -- was compared with that of the residents of Chembur and Lalbaug, the latter with high density of traffic. The survey revealed that sulphur dioxide and nitrogen dioxide levels were higher in Lalbaug. They were responsible for frequent colds and coughs, dyspnoea in residents. It concluded that, "urban subjects from more polluted zones had higher abnormalities including lower and upper respiratory tract problems, chronic heart and lung diseases with evidence of eye, nasal and skin irritation". Cancer of the lungs can be linked with benzopyrene levels which are high in Bombay because of the larger number of cars.

Table 5

Estimated Annual Doses of Respirable Suspended Particles (milligrams per individuals)

Gujarat villages	
Cook	21,000
Non cook	3,700
Traffic Police in Ahmedabad	2,600
Ahmedabad City	2,100
WHO Recommended level	210

Source : Kirk Smith, East West Centre.

Table 6

Estimated Annual Doses of Benzo(a) Pyrenes (micrograms per individual)

Gujarat villages (Cooking only)	4,200
Ahmedabad Kitchen (Cooking only)	6,100
Traffic Police in Ahmedabad	480
Ahmedabad city (Polluted areas)	180
Proposed USSR ambient standards	3.6

Source : Kirk Smith, East West Centre

### Domestic Pollution

Conventional cooking in rural India using firewood and cowdung as fuel in open chullahs with no arrangement to take away the smoke is another culprit. A study carried out by scientists for NIOH, Ahmedabad and Jyoti Solar Energy Institute at Vallabh Vidyanagar of four Gujarat villages in 1981 reveals the extent of air pollution produced by this and its ill effects mainly on women. Exposure to Total Suspended Particulates (TSP) and Benzo(a) Pyrenes (BaP) were measured. The results were shocking. Estimated annual dose of respirable suspended particulate (i.e. less than 3 millionth of a metre) in these villages were very much higher than the recommended WHO standard (see Table 5 and 6).

The average exposure to BaP which is suspected to be a carcinogen, in 3 hours in a Gujarat home, was roughly equivalent to smoking 20 packets of cigarettes per day in terms of BaP (20 nanograms per cigarette).

Its effects on the health of women can be assessed by a survey carried out over 15 years on hospital patients in Delhi suffering from Cor Pulmonale. It was found that the incidence in men and women was similar even though 75 per cent of men as compared to 10 per cent of women were smokers. In developing countries the incidence of this disease has followed the smoking pattern being more in men. In addition, age of onset in women was much lower. The authors of this study, Dr.S. Padmavati and Dr. S. Arora concluded, 'Cigarette smoking is prevalent all over India and must be a contributory cause of bronchitis in men but not in women as only 10 per cent of them smoke. The women are, however, exposed to smoky primitive fire places from childhood ...'. "From this study it appears that in Delhi, domestic air pollution is probably the cause of the higher prevalence of Cor Pulmonale in women than in men and of the early onset".

Another study in a remote mountain village also indicates a distressingly high incidence of acute respiratory infection (ARI) and was found to be the most important cause of infant mortality. Out of the total mortality rate of 490 per 1,000, 333 were due to ARI. This is about the world's highest and it is not difficult to believe that this is partially due to irritation caused by indoor, smoke-filled conditions.

Besides particulates and BaP, chullah smoke also contains carbon monoxide and formaldehyde. Carbon monoxide may worsen the already anemic condition of Indian women. Formaldehyde causes irritation in the eyes, nose and throat. It is poisonous to lung tissues and is considered to be carcinogenic. There is evidence that formaldehyde and BaP act synergistically to hasten tumor growth in animals.

### Noise Pollution

A noise level study conducted by SOCLEEN in crowded localities of Bombay in 1980, found noise levels upto 97 Decibels (Db) during Ganpati festival. The noise levels at airports measures around 90 Db. Prolonged exposure to noise above 90 Db can cause permanent deafness. Factory workers show neurological, digestive and metabolic disorders under such conditions.

### III. Habitat

Even though India is considered to be a rural country its urban population is extremely large, thanks to large scale migration from the countryside. It is important to note here that the health standards of this relatively neglected rural population and those of urban population remain nearly similar. Housing, water supply, sanitation and transport for both the urban and rural population remains insufficient and inefficient. (for water supply see Section on Water).

#### Overcrowding

Lack of housing has given rise to a large number of pavement dwellers in every city, living a life which is far from healthy. Overcrowding has become a rule not only for the poor but even the middle class in urban areas with land prices skyrocketing. Let us take a look at the situation in Bombay. 82 per cent of the current 8 million population lives in one-room abodes including slums. Half a million people live on pavements and 1.3 million live in chawls with common toilets.

In another city Kanpur, the situation is even worse.

It is estimated that two-thirds of the approximately 1.7 million population lives in one-room tenements and nearly half the city's population are slum dwellers. The effects of such overcrowding are evident on the health of the people. The Kanpur Development Authority estimates that 60 per cent of the city's children below 6 years of age are infected by TB, the highest incidence of TB in the country. A third of the slum dwellers are reported to be continuously sick according to one survey. A recent study by the Calcutta Metropolitan Development Council, of an upgraded slum showed that the incidence of TB was ten times higher than in a nearby non-slum area, viral infections two to five times greater and skin diseases twice as high.

#### Sanitation

The urban garbage -- about 20 million tonnes -- is usually left lying in the open, spreading disease. Though composting is cheap, it is not resorted to and it is eventually burnt.

Open air defaecation is a common practice. Atleast a third of urban population, well over 50 million people, do not have access to latrines.

A sewage system, being highly capital intensive, is out of reach of the majority of the people. Not more than 10 per cent of urban population has domestic connection. A strong point against sewer system is that they pollute water sources and create stinky cesspools which breed mosquitoes.

The problem of increased incidence of dengue fever raging in Indian towns has reached alarming proportions. Several epidemics have been reported from Delhi, Hardoi, Kanpur, Asansol, Calcutta, Gwalior, Sagar, Jabalpur, Jaipur, Pune and Surat. The incidence has been as high as 200 to 300 per thousand every time.

In 1982, there was an explosive outbreak of dengue fever in Delhi which affected hundreds of thousands of people in three months. In 1983, dengue again hit the city. A survey by National Institute of Communicable Diseases reported in 'India Today' showed that nearly a third of the city's population had fallen prey to the fever in the previous three months. The outbreak was found to be as a result of variety of factors such as improper urban planning, inadequate control over construction activity which blocks drainage and creates puddles, inadequate water supply which forces people to store water in overhead tanks which are seldom cleaned, discarded cans and plastic bags in which water can collect.

#### Urban Transport

While on the one hand there has been a phenomenal rise in the number of privately owned as well as public vehicles, on the other hand the commuting habits of the people have changed. This has resulted in the situation of both the road and rail transport becoming unbearable.

Public Transport - Bombay's public transport network is considered to be the best in the country. Yet its buses carry more than twice their capacity. Bombay's suburban trains carry about 4 million passengers every day. Each local train carries 4,000 persons during peak hours as against a capacity of 2,600. This adds to the problem of overcrowding and to the stresses of urban life.

Between 1961 and 1978, the number of cars (including jeeps, stationwagons) shot up by 244 per cent in Bombay and 560 per cent in Delhi. The number of buses rose by 158 per cent in Bombay and 380 per cent in Delhi. Motor vehicles generate atmospheric pollutants (see under the Section on Atmosphere). They cause noise and vibrations. Worse, they increase the risk of road accidents.

Road accidents - According to official statistics, there are about 20,000 deaths in a year due to road accidents in India, about 8 per cent of the world's total. The National Transport Policy Committee has estimated that Delhi has the highest rate of road accidents in the world with 75.5 per 10,000 vehicles. Its vehicles are the deadliest in the world with 12.5 deaths per 10,000 vehicles as compared with 1.7 for Washington D.C. and 2.7 for Los Angeles. The National Highways take an even greater toll - 57 deaths per 10,000 vehicles! Much of this can be attributed to reckless driving by bus and truck drivers.

#### IV. Agriculture

The population of India is increasing at the rate of 4.6 per cent per year and by the end of the century it is expected to touch almost a billion. This will mean that annually 230-240 million tonnes of food grains will have to be produced as against 130 million tonnes now. The ability to produce so much food grains -- on which would depend the nutritional level of our people -- would depend the health of our soil.

##### Soil Degradation

An estimated 175 million hectares -- 60 per cent of the 266 hectares of total land needing proper management -- are already degraded by water logging, salinity, and endangered by flood and recent canal irrigation schemes. Irrigation with poor drainage, seepage through unlined canals, application of excess water to crops and neglect of drainage while building railway and road embankment are the root causes of such a deplorable situation.

##### Soil Infertility

Indian soils are considered to be generally poor in plant nutrients. But depending exclusively on chemical fertilisers to enrich it can be extremely dangerous to soil fertility. Every crop removes both macronutrients like nitrogen, phosphorous and potassium (N, P and K) and micronutrients. In fact, many farmers add only nitrogen, other fertilisers being expensive. Micronutrients replacement is difficult, being out of reach of the majority of farmers. This leads to serious micronutrient deficiency. Crops from the Green Revolution area in Punjab have begun to show micronutrient deficiencies. Adding micronutrients in chemically pure forms can lead to another micronutrient deficiency. For example, in villages of Ludhiana district of Punjab, where farmers use zinc as fertiliser, zinc deficiency was replaced by copper, iron and manganese deficiency. Easier solutions of increasing use of organic manures and multiple cropping with legumes which could put micronutrients back into the soil are not being paid enough attention.

Micronutrient deficiency affects the quality of food therefore human health. Consumption of zinc deficient food can lead to retarded growth and retarded sexual development, defective wound healing and carbohydrate intolerance, according to research done by the Post Graduate Institute of Medical Sciences, Chandigarh. Also, application of zinc is shown to lead to an appreciable increase in crude proteins and carbohydrates.

##### Pesticides and Health

Nearly 60,000 tonnes of pesticides are entering the Indian environment every year. Nearly two third of these are used in agriculture. Pesticide consumption is rising at about 12 per cent per annum.

A 1965 report revealed that the accumulation of DDT in the body tissues of Indians is the highest in the world. Much of it seems to be ingested through food. Indians may be ingesting 20-40 times as much DDT as Britons.

High levels of pesticides residue have been found in vegetables, fruits and cereals in the market. Washing and cooking may reduce pesticides residues, but do not remove them totally. Even boiling removes only 35-60 per cent of organophosphorous (malathion etc.) and 20-25 per cent of organochlorine residue (DDT and BHC). Pesticides above tolerance limits can be found in cooked food, being more in a non-vegetarian meal than in a vegetarian meal.

National Institute of Nutrition, Hyderabad has reported a dramatic case of what is suspected to be due to pesticide poisoning. In 1975, health authorities of Malnad area in Karnataka reported a mysterious disease. It began with intermittent pain in the knee and hip joint hardly stand up. In the period between 1969 and 1977 over 200 people were affected by what was named Endemic Familial Arthritis of Malnad. All the affected people were poor harijans, who in time of food shortages consumed crabs caught in the rice fields; with the introduction of high yield varieties of rice which need pesticide, crabs got contaminated with parathion and endrin. The crabs even more because the land owners had stopped giving food as part of their wages. This particular disease was caused because of the susceptibility of Malnad harijans to pesticide residue due to their peculiar genetic

characteristics. Nevertheless, this shows how dangerous environmental changes could be to the poor. Also, the use of pesticide in the field means that farmers too, cannot breed fish for additional food.

According to Praful Bidwai reporting in 'Times of India', India accounts for a third or more of nearly 5,00,000 cases of pesticides poisoning estimated by WHO to occur every year in underdeveloped countries. The worst affected are agricultural and anti-malaria workers who spray and apply pesticides (see Section on Workplace).

Bhopal has brought to light another pesticide hazard, the raw materials and intermediaries manufactured and stored at the plants (see the Section on Hazardous Products).

Also, pesticides which are banned in the developed countries have been sold to the developing countries.

A survey by the Union Agricultural Ministry of markets in U.P., Bihar and M.P. found over half of the pesticides sample to be sub-standard, including 45 per cent of those cleared by the ISI. Sub-standard pesticides could be even more toxic.

##### Dams and Irrigation

Dams and irrigation are essential, it is argued, to increase the productivity of Indian agriculture. Large dams were once revered as 'modern temples of India'. All major rivers and dammed or are in the process being so. Only a fraction of hydropower potential has been realised. This indicates that in the next 20-30 years dam construction is going to be a major activity.

Recent experience has shown that these 'modern temples' have caused some severe problems to the health of the people. For example, the Narmada Basin Development programme which will involve building 329 dams costing Rs. 25,000 crores would result in the displacement of a million people, mostly tribals. The Bhopalpattanam Inchampalli project would displace around 75,000 Muria Maria and Gond tribals of Bastar. Displacement of settled communities dependent on food gathering and primitive agriculture cannot be offset by financing a rehabilitation programme. It is a question of their social health. Displaced tribals are to be resettled in an unknown area with a different language, an alien culture of individualism and competition, being forced to pursue unfamiliar occupation of modern dryland farming. Tribals depend upon forests for subsistence - hunting, collecting mahuaflowers and tendupatta, headloading dry twigs for urban homes. Uprooting them from forests and resettling them on agriculture is 'cultural ethnocide', causing grave physical, psychological and social handicaps to the settlers.

There are other problems related directly to health caused by large dams and extensive canal irrigation.

a. According to NIN Hyderabad, water seepages from the reservoir of the Nagarjunasagar dam and its canals have increased the level of subsoil water. As a result, the alkalinity of soil has increased. This in turn, has changed fluoride, calcium, molybdenum (Mb), zinc, magnesium composition of the soil. This has resulted in a suitable condition for higher intake of Mb by Sorghum plants. For people consuming sorghum as staple, this has meant higher intake of Mb and as a consequence high copper excretion leading to copper deficiency. Associated with high fluoride intake -- skeletal fluorosis has been endemic in this area -- now this has meant an acute incidence of Genu Valgum - knock knees. Apart from physical hardship this disease has led to great emotional and social stress. There has been several cases of broken families following married women developing this malady.

This problem has also emerged in fluorotic areas of Karnataka and Tamil Nadu where new dams have been built. Cases have been reported from villages around Parambikulam-Aiyar dam in Tamil Nadu and Hospet dam in Karnataka.

b. Japanese Encephalitis is a viral disease which can be fatal in 25-30 per cent of those affected. This is comparatively a new disease in India. Certain areas in Karnataka, Andhra Pradesh, West Bengal, Tamil Nadu and Goa were considered to be endemic areas. The disease is now spreading to newer areas.

The disease is carried by a species of culicine mosquito which breeds in the standing

water of paddy fields. With increase in the area of rice cultivation, multiple cropping and flooding of more land, the habitat of the mosquitoes have increased. This has led to a marked increase in their population and the incidence of Japanese Encephalitis (see Table 7).

Table 7 - Incidence of Japanese Encephalitis in India

Years	Cases	Deaths
1978	7463	2755
1979	2845	926
1980	3478	1436
1981	3894	1167
1982 (provisional)	3516	1261

Source : Ministry of Health and Family Welfare.

c. Even after pumping nearly 50 per cent of the health budget between 1950 and 1060 and achieving a dramatic reduction in the number of cases of malaria, the disease has shown resurgence. The most disturbing feature of this resurgence is the increase in the incidence of malaria caused by *Plasmodium Falciparum* which can cause cerebral malaria. While the malaria incidence decreased by 44 per cent between 1978 and 1982, the share of falciparum malaria increased from 15 per cent in 1978 to 25 per cent in 1982. Besides development of insecticide resistant mosquitoes and drug resistant organisms, environmental changes brought about by canal irrigation is a major cause of this. In a study of comparable villages in Meerut district, U.P., and Gurgaon district, Haryana, the former had six times as many malaria cases in June and nine times as many in October as did the Gurgaon villages. They also had a higher number of falciparum cases : 50 against 3. The Meerut villages were within 2 Kms of irrigation canals while Gurgaon villages were atleast 40 Kms away from them.

The study also concluded that untidy and defective canal irrigation led to water logging and the formation of puddles which were good breeding ground for mosquitoes.

Similarly, a study from Tamil Nadu shows that Sathanur reservoir and vicinity accounts for 51 per cent of all the malaria cases in the state. The heaviest concentration of cases is found in villages situated within 5 Kms of the river. Most of these villages were free from malaria for nearly 20 years.

d. Filarisis - The threat of filaria spreading has also increased. In 1963, there were 25 million at risk from filaria, by 1968 the number rose to 136 million and by 1976 to 236 million. This increase is not due only to population increases; many areas which were once free from filariasis now report cases.

Another aspect of extensive agriculture is its impact on traditional source of cooking energy - firewood. Increasingly, farmers in their pursuit of higher production are cutting down trees to bring every square inch of their land under intensive cultivation. On the other hand, replantation of trees is neglected. With whatever fuelwood available being 'exported' to cities where the demand is greater, villages are already facing an energy crisis. Other sources of cooking energy like kerosene are always in short supply. According to the FAO document "Agriculture - toward 2,000", fuelwood production is expected to fall short by 40 per cent. Many poor people will not be able to cook their food adequately. This can have serious nutritional and health consequences.

The digestibility of food will decrease and the incidence of parasites ingested with insufficiently cooked meat (and vegetables) will rise. There are reports of these happenings already in some areas.

## V. Workplace

"The health of workers in the country, be they urban or rural, organised or unorganised, housewives or clerks, has been largely ignored."

- CED, Counterfact 4

Workplace, the centre of productive activity of human society has become more and more dangerous to human health. Not that it should be necessarily so, but rapid and unplanned growth of industries in a system where labour is but a commodity to be purchased, new processes and products meant that workers face fresh risks of work-related diseases. Preventive measures are opposed by the industry as they cut into profitability.

### Industrial Workers

In a survey of 58 workers conducted in 1958, it was found that only about 5 per cent of the workers appear to be healthy. Nearly two thirds had visual defects, 43 per cent of the morbidity was related to mouth and throat and the digestive system and one third due to respiratory disorders. Malnutrition was found to be quite common. (Mitro and Banerjee, 1962).

Work place environment can become dangerous due to chemical hazards like dust, fumes, gases, vapours, acids and alkalis, solvents and other products and intermediaries; due to physical hazards like noise, temperature and humidity radiation, vibration, light and pressure; and due to biological hazards from infective agents and parasites and mechanical hazards like protruding or moving parts of machines and falling objects.

Table 8 - List of some of the harmful Contaminants and the Industries where they are likely to be encountered

1	2
Contaminant	Industry/Industrial Operations
Organophosphorus compounds (Parathion, malathion, DDVP, etc.)	Pesticides manufacture and formulation
Chlorinated hydro-carbon (DDT, BHC, Aldrin, Endrin, Heptachlor)	Pesticides manufacture and formulation
Heat	Engineering, steel and glass
Biological agents	Pharmaceutical, meat, leather, tanning
Noise	Engineering, tanning, chemical
Radiations (ultra violet and infrared)	X-ray, welding furnaces
Lead	Glass, storage battery engineering
Mercury	Caustic soda manufacture, scientific instruments manufacture, pharmaceuticals, pesticide, electrical
Carbon monoxide	Steel, chemical and engineering
Oxides of nitrogen	Fertiliser and chemical
Cyanide	Chemical and engineering
Aromatic nitro and amino compounds	Dye-stuff, pharmaceutical and chemicals
Solvents	Fertiliser, pharmaceutical, engineering, dye-stuff, organic chemicals
Benzene	Dye-stuff, steel plants, pharmaceuticals, pesticides, petrochemicals
Dust	Foundries, asbestos manufacture, ferro-manganese plant, cosmetic, rubber manufacture, steel plants, glass, cotton and textile
Hydrogen sulphide	Tanning, refineries, viscose rayon chemicals
Polychlorinated biphenyls (PCB)	Heat exchangers, transformer oils and cleansing agents
Vinyl chloride	VC and PVC manufacture

Sources: Gupta V.P., Central Labour Institute as given in Duggal, Ravi 1982.

1. Dusts - Probably the gravest of occupational diseases are caused by dusts - those generally known as Pneumoconiosis specifically referring to silicosis, asbestosis, byssinosis and other dust related chest disorders.

Silicosis - Exposure to dusts containing free silica is intense in the mining, quarrying and mineral processing industries. It is estimated that about 21 million workers are exposed to silica dust in these industries (Krishnamurthi, 1978).

Silicosis was first reported from Kolar Gold Fields in 1947. It continues to be severe even now.

A clinical and radiological examination of 7,653 workers of underground mines with 5 or more years of service revealed that incidence of silicosis was as high as 43.8 per cent (Caplan, et al 1967).

An ICMR study of 605 slate pencil workers of Mandsaur in Madhya Pradesh revealed that 80 per cent of workers were below 35 - half of male workers being under 35 and half of the female workers being under 37 years of age. No one worked or survived beyond 10 years. In Mandsaur, Multanpur and Pipalimandi villages there was hardly a man over 40 and there was not a single house without a widow. The study showed that the prevalence was highest in cutters and increased with duration of exposure. Fifty two per cent of the workers had dyspnea and 51 per cent showed positive radiological evidence of silicosis. An estimated 5,000 workers are faced with early death as there is no effective treatment.

Similar studies of mica and gold mines, reveal an equally horrible situation. According to a random survey by Central Mines Research Station, Dhanbad, 8 per cent of coal miners suffer from pneumoconiosis. This is an underestimate for even US figures are higher.

Byssinosis - Textile and fibre based industries employ 1.05 million workers and cotton mills alone 0.8 million workers. They are all at risk of getting byssinosis. Byssinosis generally affects those who have been employed for more than 10 years. Even in an advanced stage a byssinotic person cannot be distinguished from one having chronic bronchitis, on X-rays alone.

The KEM and MGM hospitals and CLI, Bombay studied three cotton mills between 1970 and 1975. The overall incidence of the disease among exposed workers was 12 per cent, it being higher in the carding section. The disease has been reported from cotton mills in Ahmedabad, Delhi, Bombay, Kanpur, Madras, Madurai and Nagpur, the incidence varying between 6 to 20 per cent.

A variant of byssinosis, bagassosis occurs when bagasse fibre or sugarcane dust is inhaled. Although the disease is transitory, it has been known to kill. In the same manner, workers engaged in hemp, sisal and flax industries suffer allergic reactions as a result of being exposed to dust.

Asbestosis - There are around twenty factories employing 7,000 workers. According to Dr. S.R.Kamat quoted in a CED report (asbestosis:dust that kills - Centre of Education and Documentation 1983) 1/3 of the workers in these factories are suffering from asbestosis! Asbestos fibres, deposited in lungs can cause pulmonary fibrosis leading to respiratory insufficiency and in more severe cases to cancer of air tubes, lungs and GI tract. The latter may strike even 5-10 years after the exposure. It is found to be more common in workers than in non-workers.

CLI found that 58 out of 850 workers of an asbestos cement unit at Faridabad, were suffering from asbestosis.

Another study by NIOH, of 800 workers of an asbestos company in Bombay revealed that 224 workers suffered from advanced stages of asbestosis while another 128 had just contracted it. In another unit in Maharashtra 40 out of 200 workers were found to suffer from this disease and 16 died of it - with lung function varying from 10 to 25 per cent.

Families of the workers are also vulnerable because the workers carry home fibres sticking to their clothes. U.S. government health officials estimated in 1978 that in U.S. alone 50,000 new cases of cancer would occur annually for 30 more years as a result of past exposure to asbestos.

Conditions in asbestos mines are even worse. Some 1,500 tribals are employed in India's largest mines at Roro - owned by the Birlas. They crouch for hours in dingy underground tunnels. Moreover, the fine asbestos dust is dumped in the open, exposing 50 tribal villages in the neighbourhood.

All these diseases are notifiable under the Factories Act. However, as N.L. Gadkari, former Chief Inspector of Factories in Maharashtra, underlines "the moment a disease becomes notifiable, it just disappears".

2. Chemicals - The growth of the Indian chemical industry has been phenomenal (see Table 9). Although the adverse effects of exposure to chemicals are well known only in few studies has a strict cause-effect relationship been established. Nor is it clear how far the background health status of the worker exacerbates toxic effects of chemicals.

Table 9 - Growth of Industries dealing with Toxic Chemicals and Generating Toxic and Hazardous Wastes

	Production (thousand tonnes)		
	1950	1970	1980
Pesticides	NA	3.00	40.68
Fertilizers (nitrogenous and phosphatic)	18	10.59	30.05
Dyes and pigments	NA	13.55	30.85
Pharmaceuticals	0.15*	1.79	5.07
Organic chemicals including petro chemicals	200	17100	24100
Steel (ingots)	1500	6500	8000
Non ferrous metals			
Copper	NA	9.30	18.80
Lead	NA	1.86	11.40
Zinc	NA	23.41	52.70
Caustic soda	11	304	407

NA = Not available

\* Value for 1952.

Source : B.B.Sunderesan, P.V.R.Subrahmanyam and A.D.Bhade, An overview of toxic and hazardous waste in India, Industry and Environment, Special Issue 1983.

Inhalation of fumes, dusts, direct contact with acid, alkalis and other irritants as well as inhalants, and ingestion of toxic metals like lead and mercury cause severe morbidity amongst workers of chemical industries. Extremely little is known about the incidence of diseases caused by chemicals.

For instance, a large number of workers are employed in industries manufacturing lead pipes, paints and other products where they are exposed to lead. But even in a developed state like Maharashtra, not a single case of lead poisoning of a worker was reported between 1971-1983. CLI studied 360 workers of a lead acid storage battery unit in Bombay and found that around 9 per cent suffered lead poisoning and two third were exposed to air borne lead in excess of the Threshold Limit Values (TLVs).

In another study conducted by CLI of three caustic plants in Bombay where cases of mercury poisoning were reported, about 6.5 per cent of the exposed workers had bleeding/sore and receding gums as compared to none of the control workers (Gupta and Singh).

A survey conducted by NIOH in 1978-79 of an insecticide factory in Delhi revealed that 73 per cent of the exposed workers manifested toxicity symptoms.

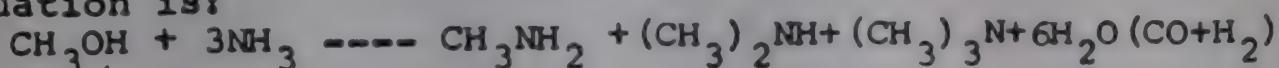
The situation is further complicated by inappropriate and biased studies done by government sponsored agencies. In June 1983, Padmanabhan V.T. conducted a survey of the workers of Gwalior Rayon Silk Mills (GRASIM) factory at Nagda in Madhya Pradesh. According to the survey, 15 workers out of 5,000 had died of work related causes in 1982 and a further 27 were suffering from such ailments. The toxic agents identified are carbon disulphide and sulphur dioxide. A study conducted by CLI, however showed that the atmospheric concentration of these substances were far below their respective TLVs, and that the health status of the exposed workers was not significantly different from that of the controls. As Padmanabhan points out in his report, the major shortcoming of such studies is its dependence on a general TLV. In poor countries where the health status of the worker is already low, this is not a sufficient precaution. A medico environmental study is more reliable. Moreover, according to him, the sample studied by CLI did not include temporary workers and dismissed/retired employees.

It should be remembered that these very industries are the major cause of atmosphere and water pollution and thus, a threat to the people living in the surrounding area. GRASIM factory at Mavoor, Kerala has also come under criticism for polluting the Chaliyar river, killing thousands of fishes and making hundreds of hectares of land uncultivable.

THE PRODUCTION OF PESTICIDES & AIR POLLUTION  
a case study of a methylamine plant  
by Madhusudan G. Rao, Env.Engineer.

The production of pesticides necessarily involves the manufacture, storage and process of a wide range of hazardous substances and byproducts, many of them in an unstable or gaseous state. This piece examines one such chemical - monomethylamine (an intermediary for MIC used to manufacture carbaryl at Bhopal).

Methylamine is produced by means of a reaction between ammonia and methanol at an elevated temperature. A mixture of mono, di and tri - methylamines is formed. These are then separated and purified by distillation. The chemical equation is:



The process can be broken up into three stages.

1. **Synthesis:** Here, measured streams of methanol and ammonia are fed continuously through vaporizers and heat exchangers into a converter unit packed with an  $\text{Al}_2\text{O}_3 - \text{SiO}_2$  catalyst. In the converter, the above mentioned reaction occurs. The operating pressure is around 300 psi and the temperature is  $450^\circ\text{C}$ .
2. **Distillation:** The distillation is done in four columns and associated condensers and reboilers, hooked up in series. Since the excess ammonia in the crude mixture from the converter is very volatile, it is separated first in the overhead column and returned for refeeding to the synthesis section. Trimethylamine, dimethylamine and monomethylamine are then separated in that order in these distillation columns.
3. **Production handling:** The products are stored in storage tanks provided with relief valves.

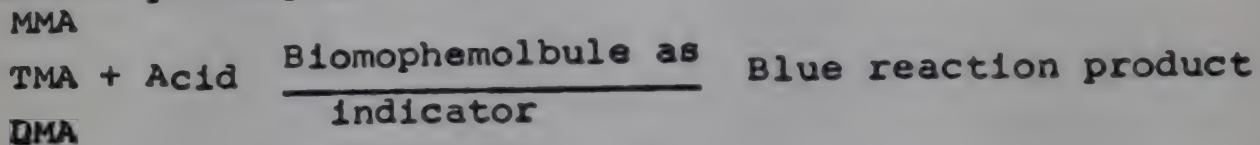
The obnoxious gases/vapours emanating from the methylamine plant must be destroyed in order to avoid air pollution. These vapours are released and neutralized through the vent absorber and blow-outs through relief valves in the distillation columns and storage tanks.

- a) **Vent Absorber:** The typical percentage analysis of the gas neutralized here is as follows:  $\text{H}_2$  (28.66%),  $\text{A}^*\text{O}_2$  (1.07%),  $\text{N}_2$  (3.31%),  $\text{CH}_4$  (4.94%),  $\text{CO}$  (15.31%),  $\text{NH}_3$  (33.20%), MMA (0.37%), DMA (0.51%), TMA (8.96%),  $\text{H}_2\text{O}$  (3.67%). The rate of flow in the absorber is usually very slow.
- b) The Relief Valves of the distillation columns, batch tanks, recycle tanks and the ammonia-feed tanks may open under high pressure if the temperature of the cooling

water in the condenser is not maintained. The gases thus released need to be burnt before being vented. (Scrubbing of gases is not desirable as this will create liquid effluents, which are difficult to dispose of.)

c) The relief valves of the storage tanks which hold anhydrous amines need to be isolated since it may malfunction and release gaseous amines unexpectedly. The amines from these sources should be destroyed by flaring which can be done by means of a flare system for which sophisticated designs are now available.

Air monitoring : There are no ambient air quality standards for methylamine. The TLV for monomethylamine is 10 ppm and the approximate threshold of smell is 0.02 ppm. There also appears to be no method to analyse methylamines separately as MMA, DMA, TMA at very low concentrations. They (in concentrations between 5 and 60 ppm) can however be detected together by using a Drager tube as per the following reaction principle:



Errors in this method can however be as large as 50%. Besides ammonia, hydroxine, diethylamine, dimethylamine, methydiethylamine, monomethylamine and monoethylamine are indicated with approximately the same accuracy as trimethylamine.

### Conclusions

1. Flaring or destruction of amines in a catalytic combustor are convenient ways of disposal.
2. Defective relief valves can lead to dangerous situations as large amounts of methylamine could be released. Hence constant inspection of relief valves to check for accumulation of dust etc. is essential. Regular valve calibration should also be carried out. It is important to maintain proper temperature and pressure to avoid valve blowouts.
3. Methylamines should be stored in horizontal tanks, so that safety devices like relief valves are accessible. The inspection of the tank also becomes easier.
4. There are no ambient air quality standards for methylamine.
5. No reliable method has been established to measure the different amines at low concentrations.
6. The DRAGER tube method with its inherent disadvantages is a quick method for measuring workplace concentrations.

Table 10 - Contaminants Level in some Industrial Work Environments

Industry	Pollutant	Pollutant levels *	Permissible standards
Sulphuric acid plant	Sulphur dioxide	12.26-35.0 ppm	10 ppm
Steel Plant	Dust containing free silica (Foundry)	3-1075 mppcf	5 mppcf **
	Refractory dust	2-17 mppcf	20 mppcf
	Carbon monoxide (blast furnace)	20-200 ppm	100 ppm
	Aromatic hydrocarbons benzene	20-50 ppm	35 ppm
A chemical factory manufacturing sodium carbonate and ammonium chloride	Ammonia	34-107 ppm	
		40-172 ppm	
		204-430 ppm	
		75-1240 ppm	50 ppm
		187-860 ppm	
Chemical work	Oxides of nitrogen (Nitric acid plant)	11-31 ppm	5 ppm
	Chromic acid dust (Chromic acid plant)	0.5-2.1 mg/m <sup>3</sup>	0.1 mg/m <sup>3</sup>
	Bauxite dust	8-198 mppcf	50 mppcf
Blending of pesticides	Soapstone dust	33-134 mppcf	20 mppcf
	DDT	15.3-17.8 mg/m <sup>3</sup>	1.0 mg/m <sup>3</sup>
	BHC	25.2-29.8 mg/m <sup>3</sup>	0.5 mg/m <sup>3</sup>
Engineering Industry (tool room)	Isophorone	296 mg/m <sup>3</sup>	55 mg/m <sup>3</sup>
Pharmaceutical laboratory and works	Talc dust	60-90 mppcf	20 mppcf
	NIBK	408-792 mg/m <sup>3</sup>	410 mg/m <sup>3</sup>
	Ethyl alcohol	482-7812 mg/m <sup>3</sup>	1900 mg/m <sup>3</sup>
Heavy Engineering factory manufacturing electrical equipments	Iron compound dust	0.67-5.58 mg/m <sup>3</sup>	1 mg/m <sup>3</sup>
	Alumina dust	15.0-32.4 mppcf	30 mppcf
	Silicone	37.1-4156.6 mppcf	30 mppcf
	Coal dust	28.0-35.3 mppcf	
	Iron dust	17.9-51.5 mppcf	30 mppcf
	Sand bearing free silica	23.5-101 mppcf	6 mppcf
	Asbestos	847.2-1517.9 fibres/cc	
	Oil mist	4-158 mg/m <sup>3</sup>	5 mg/m <sup>3</sup>
Noise	Noise	87-120 db	85 db

\* Range of Means (minimum and maximum values)

\*\* Million parts per cubic feet.

Source : Gupta, V.P., Central Labour Institute, Bombay as given in Duggal, Ravi, 1982.

Table 11 - Selected list of Toxic Chemicals for Control

	Use	Hazard
Acrylonitrile	Acrylic fibres/synthetic rubber plastics	Highly toxic/carcinogenic/teratogenic
Arsenic	Pesticides/Unani medicines/glass	Toxic/dermatitis/muscular paralysis/damage to liver and kidney/possibly carcinogenic and teratogenic.
Asbestos	Roofing/insulation/air-conditioning conduits/plastics/fibres/paper	Carcinogenic to workers and even family members.
Benzene	Octane number of gasoline/manufacture of many chemicals.	Leukemia/cromosomal damage in exposed workers/behavioural changes.
Beryllium	Aerospace industry/ceramic parts/household appliances	Fatal lung disease/heart and lung toxicity.
Cadmium	Electroplating/plastics/pigments/superphosphate fertilisers.	Kidney damage/emphysema/possibly carcinogenic teratogenic and mutagenic.
Chlorinated organics (DDT, BHC, etc.)	Pesticides/Fumigant	Depression of central nervous system/possibly carcinogenic.
Chromates	Tanning/paints/pigments/corrosion inhibition/fungicides	Skin ulcers/kidney inflammation/possibly carcinogenic/toxic to fish.
Lead	Pipes/storage batteries/paints/printing/plastics/gasoline additive.	Intoxicant/neurotoxin/affects blood system.
Manganese	Mining/welding/dry cell battery/ferro-manganese.	Nervous damage/damage to reproductive system.
Mercury	Chloralkali cells/fungicides/pharmaceuticals.	Nervous damage/kidney damage.
Polychlorobiphenyls	Transformers/insulation of electricity.	Possibly carcinogenic/nerve, skin and liver damage.
Sulphur dioxide	Sugar/bleeding agent/pollution from coal based power stations.	Irritation to eyes and respiratory system/damage to plants and monuments.
Urea	Fertiliser.	Bronchial problems/kidney damage.
Vinyl chloride	Plastics/organic compounds synthesis	Systematically toxic/carcinogenic.

Source: C.R. Krishnamurthi, Toxic Chemicals, In State of the Environment: Some Aspects, National Committee on Environmental Planning, New Delhi.

**3. Physical Agents** - Physical agents have received scant attention in the working environment.

Ultra violet radiation emitted during arc welding can damage eyes and skin. Visible light can affect health in two ways: Poor illumination can lead to accidents, glare can cause eye fatigue and visual interference. Foundry workers handling molten metals are exposed to infrared rays, which can cause eye damage besides increasing body heat. Ionising radiation is a very serious hazard. X-rays are being used for radiography of castings and welds and fluoroscopy. Beta-rays are being used for elimination of static electricity and gauging material thickness. Radio active isotopes too, are used extensively. Exposures to high level of ionising radiation can cause severe burns, blood dyscrasias, gastro-intestinal symptoms, cataract, cancer and genetic mutations. Nuclear thermal plants, nuclear reactors, heavy water complexes are particularly notorious for their serious implications to the health of their workers. (see Section on Hazards of Nuclear Energy).

Noise is a serious threat to the psychological and physical health of a worker employed in boiler making, aircrafts, motor garage, blacksmithy, weaving industry, rivetting, blasting and metal working trades, rock drilling and lathe operating. Besides causing permanent deafness and rupture of the ear drum, the worker may suffer from psychological problems. According to Industrial Toxicological Research Centre (ITRC), harmful effects of noise include increased annoyance, mental tension, irritability and emotional disturbances at home and at work. In an overview of health hazards of textile industry from 1925 to 1981, ITRC observes that greater circulatory, heart and equilibrium problems were found in textile workers, working in a noisy environment. Textile mills are one of the noisiest places in the country. The level in a large weaving section ranges from 100-105 Db and continuous exposure to such high levels of noise can cause permanent hearing loss.

Exposures to high temperature can upset the homeostatic balance mechanism in the body and can cause circulatory disturbance and heat exhaustion. Furnace and foundry workers, metal casters, iron and glass workers, miners and those working in the steam press shops in rubber industry are exposed to this hazard, since a furnace is used in a variety of industries including coal tar melting for road laying purposes, and steam engine, metal and metal based industries. High temperature can also cause apathy, lassitude and sleeplessness.

Similar effects are also produced at lower temperature but in a humid atmosphere with low wind velocity.

Vibration, shocks and abnormal air pressure that can cause severe damage to the circulatory and nervous system have received scant attention.

Hazardous biological agents consist of bacteria, fungi and parasites. In fact, almost any infectious disease may be contracted during employment. This is especially true for medical personnel. Besides doctors, nurses and laboratory workers, other workers exposed to these hazards are tanners, veterinarians, slaughter house employees, zoo and circus attendants, agricultural and municipal workers.

**Accidents** - According to official statistics during 1965-75, over 5,000 workers were killed and 2.5 lakh injured in industrial accidents. In 1979, 64 out of every 1,000 workers in the country were injured at work.

Between 1951 and 1981, average daily employment in factories increased by 120 per cent while the number of fatal accidents by 225 per cent and other injuries by 393 per cent. The textile industry which employs nearly a quarter of the total workforce — the largest single employer — registers most accidents — 127 per 1,000 employees in 1979 as against 58 in metal industry. A CLI study of accidents in the engineering industry revealed the shocking rate of 277 per thousand. Furthermore, six out of ten accidents were caused by unsafe working conditions. Textile, metal and engineering industry and mines account for the major proportion of industrial accidents.

Workers employed in small units are particularly vulnerable to accidents. To cite a few cases, 32 people died in a blow-up in a firework factory at Sivakasi, 48 people died in a Surat Silk Mills collapse and a fire consumed 9 lives in a plastic toy unit in Delhi.

Serious injuries are fewer in mines. In 1979, there were 2,853 serious injuries of which 2,122 were from collieries alone. Many mine disasters have been reported in the past, the worst of them being the Chasnala disaster. Part of the mine collapsed in 1975, burying atleast 430 miners under tonnes of water. Explosions caused

by careless handling of fire - lit cigarettes, explosives etc. are the chief causes. In 1958, an explosion killed around 200 people in a colliery in Raniganj. In 1976, 43 coal miners lost their lives in another explosion in Sudam-dih, caused by methane. According to the Coal Miners Officers Association, there are 99 unsafe coal mines in the country where operations without measures can only spell disaster.

The plight of quarry workers seldom finds official mention. A study of a single stone quarry showed that 45 workers had died in such accidents, between 1977 and 1983. Around 150 workers suffered grievous hurt during the same period.

Accidents and minor injuries occur quite frequently at construction sites but go unreported. As buildings rise taller the accident rate also goes higher. Moreover, use of sub-standard material adds to the menace. In one instance 19 workers were hurt when a flyover under construction collapsed in Delhi, allegedly in a bid to hasten its construction before the Asiad.

Workers at dam sites are also prone to serious accidents. Nine workers were buried when a huge mound of earth caved in at Kanjhari, Orissa in 1982. In Ramganga dam in U.P., a local newspaper reported 88 deaths and 101 injuries during its construction.

Unfortunately, the official data records only 20 per cent of the total accidents because it covers only those factories coming under the Factories Act. It also excludes one in three factories not reporting accidents. Accidents involving contract and casual labourers go unreported. In one instance, these formed a quarter of the workforce of a large iron & steel factory, but suffered three and half times more accidents than permanent employees.

Thus, the statistics cover only 3.3 per cent of total workforce of 230 million. In the organised sector, fatal accidents kill 16 workers out of every one lakh workers. If the same rate is applied to project the estimate of fatalities in organised, unorganised and agricultural sectors, according to a researcher from the Indian Institute of Technology, it would be in the region of one lakh deaths per year!

**Psychological impairment as an occupational hazard** - Often psychological symptoms are early warnings of serious physiological problems. Also different dusts and chemicals can affect a workers' behaviour. The earliest study on manganese workers in 1958 revealed that workers suffered from a wide variety of psychological disturbances including impotence, irritability, insomnia, bad memory and depression. CLI reported in early fifties that 7 out of 10 workers in lead storage battery units were exposed to levels higher than permissible and complained of a variety of mental symptoms. In a survey conducted in 1980 by CLI of the same units, it was found that 375 workers suffered from inability to react to pictures.

Two researchers have studied the chronic exposure of pesticides for 11 years. As many as 43 per cent complained of anxiety, sleeplessness and depression, 12 per cent reported that such symptoms worsened after spraying.

Certain sections of the working population including women in domestic work have special problems.

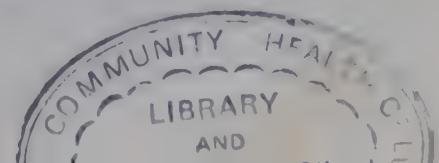
#### Transport Workers

Transport workers -- loaders at dock, railway workers, bus and truck drivers and their assistants -- face several risks.

Dock workers are particularly vulnerable because they often handle dangerous substances without being aware of their toxicity. Heavy load itself is a major occupational hazard. A survey conducted in 1980 by CLI revealed that loaders suffered stomach and back complaints. Damage to the vertebral column and the cardiovascular system was the biggest threat.

Railway workers face additional risks of accidents - there were around 300 fatal cases a year between 1962 and 1976 due to heavy work schedule, frequent night shifts, machine over use etc.

Bus and truck drivers and their assistants are the most vulnerable groups of workers as shown by a very high rate of road accidents (see Section on Transport and Habitat). While human error is usually to be blamed, factors like poor maintenance of the vehicles and bonuses paid to truck drivers for extra fast delivery of goods contribute substantially to the high accident rate.



### Farm workers

An official estimate states that about 1,000 farmers are injured throughout the country during the wheat harvesting season. The chief culprit is the thresher. Safdar Jung hospital, Delhi admitted 2,000 such patients between 1979-81 and 117 returned without a limb. According to a survey by Dr. S.R. Verma & S.R. Bhatia, Punjab Agriculture University, more than 10,000 people working on threshers have had their hands chopped. A recent survey by Department of Agriculture, Haryana showed that the overall rate was 25 accidents per thousand machines. A report in Indian Express points out that every year about 50,000 threshers are added to estimated 8 million already working.

Another major farm hazard arises from the increasing use of fertilizer and pesticides. While banned in the West, certain pesticides continue to be used in ever increasing amount, but the safety precautions to be taken by the sprayer are non-existent. To make matters worse, aerial spraying is being resorted to in certain areas. There is virtually no mechanism to study the severity and intensity of hazards in a sector in which the largest chunk of the working population of the country is employed.

### Women

This is one area of occupational health where virtually no information is available. Besides working at home, many women work in factories and on farms.

Women at home - Women work longer than men. A few available studies prove this. In Pura in Karnataka, women contribute 46 per cent of human hours spent in the village on agriculture, industry and domestic work, men 37 per cent and children 17 per cent. On domestic activities alone women contributed half the total time spent.

In households chiefly women and children spent nearly 7.5 to 10 hours a day on fetching fuel and water and cooking. A study from western U.P. shows that even pregnant women have a 14-16 hour working day. This over-work coupled with higher rate of malnutrition in women means higher morbidity and mortality in women.

Collecting firewood is a backbreaking and hazardous task. In Pura, women spend, an average of 2.51 hours daily and made 172 trips in a year, each being 8.54 Kms long. Women also spend long time fetching water varying from 1.4 hours per household per day in Pura to 3.9 hours in a village in eastern U.P.

Areas where no information is available include injuries during domestic work: knife injuries, burns, electric shocks, toxic effects of products used mainly by women - household pesticide, detergents etc. and psychological problems of domestic work.

Women at work - Women are often employed in low paying, menial tasks in the unorganised sector - lace making, beedi rolling, rag picking, headloading, assembling of electronic equipment, capsule filling in pharmaceuticals etc. In many of these areas, there exists no documented information on health hazards.

In a study of women working in a pharmaceutical factory, women were found to be handling pharmaceuticals without proper protection and thus were found to be prone to adverse reaction and sensitisation (Gotoskar et al).

Many women, especially tribal women carry wood on their heads to the urban market. It is possible that atleast 2 to 3 million people, mostly women may be headloading today. Collecting firewood could be hazardous because it means negotiating rough terrain often several times in a day. A study by Self Employed Womens' Association, Ahmedabad of the headloaders in the Girnar forest shows that around 58 per cent of women are hurt by axes while cutting trees.

### VI. Hazardous Products

With the growth of industries, potentially toxic products and hazardous wastes have also grown especially in pesticides, dyes and pigments, organic chemicals, fertiliser, ferrous and non-ferrous metal chloralkali manufacture,etc.

Storage areas for waste from phosphatic fertiliser plants near factory sides are now getting exhausted and the lagoons containing waste slurry are overflowing into surface water and are leaching into ground water.

Fly ash from thermal power plants containing toxic elements like zinc, barium, copper, arsenic, vanadium, manganese etc. can overflow into surface water and can

### leach into ground water.

Pesticide industry, besides producing extremely hazardous products (see Section on Agriculture) also produce highly toxic wastes. Even the heated sludge containing waste products has a high amount of pesticides.

The sludge is dumped on low lying areas and the leachate pollutes both ground and surface water. Moreover very little is known about the processes involved in the manufacture of pesticides, their raw materials and the intermediaries used. It is reported recently that a leading pesticide unit in Bombay is manufacturing ethylene dibromide described as the "most potent cancer causing substance found in the animal test programme", by the National Cancer Institute in U.S. A plan to manufacture glyphosphate is being put up. They intend to use chloromethyl phosphonic acid (CPA) in the process; CPA is a chemical used in chemical warfare.

Petrochemicals, coal and ethanol based organic chemical complexes are the most potent sources of established mutagenic and carcinogenic chemicals. Acrylonitrile, acrylamide and vinyl chloride used in the manufacture of plastics polymers, artificial fibres, synthetic dyes, drugs & pharmaceuticals and pesticides are the major hazardous products.

The chloralkali industry used mercury, most of which finds its way into surface and ground water through toxic waste water. Minamata disease has yet to be reported in India but the mercury load of water sources near rayon, paper and chloralkali industry is rising (see the Section on Water).

Even small industries deal with toxic substances. For instance, for every tonne of hides and skins processed 3.5 to 4.5 Kg. of trivalent chromium gets discharged as waste. Units dealing with electroplating, bangle making, processing of natural fibres and goldsmithy too, use toxic chemicals.

### Accidents during Storage and Transport

A massive explosion of the type that occurred in Bhopal is rare only in terms of scale. Several accidents have occurred during storage and transport of chemicals but have received attention only after the Bhopal disaster.

A major accident involving stored benzene hexachloride and sodium occurred in Delhi in 1984; nobody was killed but two children fainted and two labourers received burn injuries.

After Bhopal several accidents have been reported. Three people died and three fell critically ill, when a tanker carrying ammonia collided with an empty oil tanker near Chandigarh. A week later, a girl died and three were injured when a tanker carrying sulphuric acid turned turtle near Vadodara, Gujarat. About a fortnight later, 47 people became ill when large quantities of chlorine were released accidentally in Cochin. In early January this year, gas leaked from sodium hydrosulphide stored in a warehouse near Jabalpur, affecting 100 people. In August this year, chlorine gas leaked from a chemical factory in Chembur killing one and affecting thousands of people.

### VII. Hazards Of Nuclear Energy

Today, India has six nuclear reactors, five heavy water plants, two nuclear waste reprocessing plants and one nuclear fuel complex, all handling radioactive material at various stages. Besides the ever present risk of an accident the effects of which are unestimable, the hazards of chronic exposure to varying levels of radiation are immense.

In the uranium mines of Bihar, the ore is processed to obtain a concentrate of oxides. The mill tailings are decanted and the fluid is channelled into a nearby nullah; to this is also added the discharge from the mine, containing uranium, radium and manganese. When the effluent reaches a nearby river, the concentration of the radioactive material is very high. Even at sites 10 Km. away from the plant, grass and milk samples show presence of radium.

The workers employed in the mine face hazards of inhalation of radon gas and its decay products. These irradiate the respiratory tract and the lungs. As many 16 out of every 100 miners have a high probability of dying from cancer, especially if they smoke, despite precautions. Given the low nutritional status in the backward region of Bihar the harm caused is likely to be ever greater.

Though as yet no major disaster has occurred in India,

situations of near-disasters have been many. In a nuclear reactor even a minor accident can spell a serious disaster. The seriousness lies in the fact that "a routine check, some careless decisions, mistiming, lack of precision and slightly longer delay, within minutes can transform a simple matter to a nerve shattering, horrifying crises, the magnitude of which, frankly, has no precedence yet".

The possibilities of an accident has increased several fold because often temporarily, untrained workers are used in such situations. Since they do not wear badges, nor do they undergo routine examination nobody knows the exact amount of radioactivity they receive but it must be very high. For example, till end of 1982, a cumulative total of 10,806 temporary workers had been used in the Tarapur Atomic Power Station (TAPS), and they received a cumulative dose over 1,928 rems. And all the reactors in India have high radiation fields, the worst being Tarapur. The total human rem exposure has increased from an average of under 500 rems a year in the beginning to the current 5,000 rems. The average dose at the TAPS has increased from 117 millirem in 1969 to 4,069 millirem in 1982 - a 35 fold increase. Tarapur's collective worker-dose per reactor is 270 per cent higher than the U.S. level which itself is considered too high. Following a conservative estimate that 10,000 rems cause 6 cancers, radiation at TAPS can be expected to have caused 27 cancers. And for each additional year, it will 'claim' 2 to 3 lives and it has ten more years to go.

There is hardly any study of the effects of these nuclear plants on the health of the population living in surrounding areas though in the U.S., several studies have shown evidence of carcinogenicity of low-level radiation. Following this the U.S. Environmental Protection Agency has reduced recommended exposures for people living near a nuclear site to 25 millirem from 500 millirem a year. What is surprising is that the recommended exposure for the public and the occupational work, differs by a factor as high as 200. In fact, there is evidence that a background natural radiation from thorium contained in sands of coastal Kerala, of the order of 1.5 rems to 3 rems per annum, much less than the levels considered safe by the nuclear industry, can cause genetic damage and cancer.

Another source -- posing an even bigger problem -- of hazards of nuclear energy is nuclear waste. About 20,000 tonnes of this toxic waste material exists in the world. This is a grave liability because the half lives of many of the radioactive wastes run upto 20,000 years. India is expected to generate upto 1,07,000 m<sup>3</sup> of low level waste concentrate and 8,000 m<sup>3</sup> of high level waste. As yet no 'safe' mode of storing -- that is what disposal of radioactive waste ultimately means -- these wastes has been found. And 'safety' here means keeping it safe from floods, earthquakes, aerial bombing and sabotage for thousands of years.

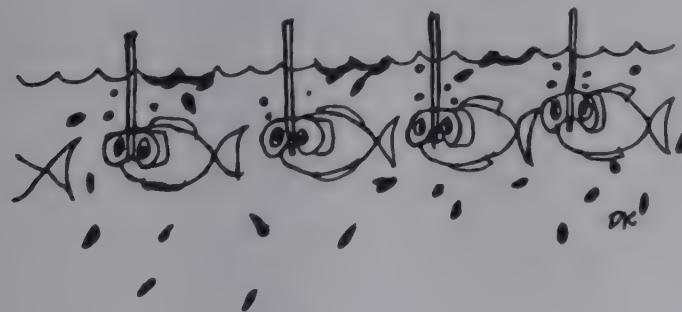
Finally, presence of nuclear 'facilities' in today's world fraught with nation state rivalries, superpower arms race and other international tensions means presence of raw materials for a nuclear war. The potential hazard to human health of such a contingency is total extinction of the human race -- probably of all living matter -- from the face of the earth.

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## GLOSSARY

1. BOD Biological Oxygen Demand, is the term used to measure the amount of oxygen required to sustain the aerobes and protozoa in the river water.
2. COD Chemical Oxygen Demand is the term used to measure the amount of oxygen used up to convert organic wastes into inorganic materials.
3. Cor Pulmonale is the term denoting the effect on the heart of chronic lung diseases resulting in the accumulation of blood in the right side of the heart leading to right ventricular failure.
4. HALF-LIFE is a measure of radioactive decay. It is the period of time it takes for a radio-active element to be reduced by a half.
5. MPN Most Probable Number is the term used to measure the coliform and fecal coliform bacteria that accompany the solid wastes discharged into water.
6. Rem. Roentgen Equivalent Man is the measure of exposure to radiation. A rem is the number of Radiation Absorbed Doses (Rads) absorbed by a target multiplied by the relative biological effectiveness of the particular type of radiation involved (alpha, beta or gamma). Low Level radiation is measured in millirem (one thousandth of a rem).  
A Rad is expressed as the amount of energy transferred to a cell as a result of radiation.



Note: Unless mentioned otherwise, all tables, statistics, case studies, reports and quotes are from The State of India's Environment 1982, and The State of India's Environment 1984-85, Centre for Science and Environment, New Delhi.

Pesticides are hazardous to human health. But these hazards affect different sections of the society in a varied manner and intensity. For those workers associated with the manufacture and preparation of these chemicals, as also for the agricultural workers who are associated with the application or the use of these pesticides, it is mainly a question of exposure to high concentrations for shorter periods. On the other hand the general population exposed to pesticide polluted atmosphere, water and food, the question is of prolonged exposure to lower levels of pesticides. There is yet another section of people - the workers who are associated with 'pest control services' (household pest eradication) and those involved with pesticide use in vector control (malaria eradication etc.). In their case, the handling of high pesticide concentration day in and day out leaves them open to prolonged exposures to high levels of pesticides.

It is plain and obvious that each of the above cases must be separately examined to appreciate the implications of pesticide hazards to our health. Simultaneously it is also necessary to investigate (a) the safety measures (b) the role and effort of the governing authorities to minimize pesticide hazards (c) the legislative measures to ensure these, and finally (d) alternatives to the use of pesticides.

This is a sizeable exercise entailing diverse expertise and considerable information. Unfortunately this write-up is deficient on both counts. The little that has been available is put in the following pages.

#### Pesticide Groups And Toxic Effects

A wide spectrum of pesticides have been employed hopefully, to rid pests of various sorts. These are fungicides, insecticides, acaricides, nematicides, molluscicides, rodenticides and herbicides, and they range from simple inorganic compounds like zinc phosphide to complex organic molecules like paraquat (1,1' - dimethyl - 4,4' - bipyridinium). Nonetheless the most frequently used ones can be classified into 3 or 4 groups in order to facilitate discussion on the toxicity of these pesticides. The major pesticides can be grouped under (a) Organochlorine compounds, (b) Organophosphorus compounds, (c) Carbamates and (d) Dinitrophenols.

Some better known pesticides of each group are:

##### Organochlorine compounds

Aldrin; DDT (dicophane); dieldrin; eldrin; HCH (also known as BHC ... benzene hexachloride); HOED; HHD; heptachlor; TDE.

##### Organophosphorous compounds

Dioxathion; endothion; malathion; metriphonate; dinon; fenthion; TEPP (ethyl pyrophosphate).

##### Carbamates

Aldicarb; carbaryl; zineb.

##### Dinitrophenols

DNC (dinitrocresol); dinocap.

(Note: Extensive list in each group is available in the addendum).

#### Toxic effects of Chlorinated Insecticides

Chlorinated insecticides produce symptoms consistent with central nervous system stimulation. They may be absorbed through the respiratory and gastro-intestinal tracts or through the skin. Whatever its route, the absorption is facilitated by oily substances. Poisoning has occurred through accidental ingestion, inhalation or percutaneous absorption of various preparations containing the substances.

Symptoms of acute poisoning include vomiting and diarrhoea, paraesthesia (abnormal spontaneous sensation such as burning, pricking, tickling or tingling), excitement, giddiness and fatigue, followed by tremors, convulsions, coma and possibly pulmonary oedema. Respiration may be accelerated initially and later depressed. Symptoms may be complicated by the effects of solvent.

Early symptoms of chronic poisoning are headache, loss of appetite, muscular weakness, fine tremors, and apprehensive mental state. Complete recovery usually occurs, if further contact with insecticide is avoided. Occasionally, liver degeneration may occur with haematological changes leading to anaemia and leucocytosis. Kidney damage may also occur.

The severity, latency and persistence of toxic effects vary with different chlorinated insecticides due, among other factors, to variation in their persistence after application, their rates of absorption, their storage in the body tissues and their metabolism.

Chlorinated insecticides have been reported to enhance the metabolism of steroid hormones such as testosterone and some drugs which are metabolized in the liver, including some barbiturates and phenylbutazone. (This is attributed to the action of these insecticides on the microsomal system).

The chlorinated insecticides are only slowly degraded and they may persist long beyond the period for which they are required to be effective. Growing concern has been expressed on the widespread and cumulative contamination of the environment with such persistent substances, particularly as the contamination is not limited to the area where the insecticide was used but occurs in the environment as a whole.

Residues of chlorinated insecticides are present in butter, cheese, eggs and meat particularly animal fat. Smaller amounts are also found in cereals, fish, fruits, milk etc.

Like always, the literature is full of conflicting reports. Whilst a report by the Advisory Committee on Pesticides and other Toxic Chemicals, London found "no proof that dicophane caused injury while stored in the fat of human beings or animals, and dicophane and dieldrin could not be condemned as presenting a carcinogenic hazard to man", L.L. Comar et al (Archs envir H Hlth 1968, 17, 306, per J Am Med Assn, 1968, 205, A167) found in 44 post mortems, the highest concentration of dicophane and other chlorinated insecticides was found in patients with emaciation carcinoma, or focal or generalised liver disorders.

#### Toxic effects of Organophosphorus Insecticides

Organophosphorus insecticides are potent cholinesterase inhibitors, owing muscarinic, nicotinic and central nervous system effects mainly to acetylcholine accumulation.

They are much less persistent than the chlorinated insecticides but are usually many times more toxic. They are readily absorbed through the skin and the respiratory and gastro-intestinal tracts. There are many recorded instances of poisoning.

Exposure to the vapour or to particulate material may cause miosis (contraction of the pupil), sometimes unequal hyperaemia (the presence of an increased amount of blood in a part) of conjunctiva, dimness of vision, rhinorrhoea, frontal headache, bronchoconstriction, increased bronchial secretion, cough, nausea, vomiting and fasciculation (involuntary contraction or twichings) and sweating at the size of contact.

After absorption or ingestion, side-effects, which may be delayed for upto 12 hours, may include anorexia, abdominal cramps, diarrhoea, involuntary defaecation or urination, weakness, dyspnoea, lachrymation, increased salivation and sweating, bradycardia, cyanosis and muscular twitching of the eyelids, tongue, face and neck, possibly progressing to convulsions. Central nervous system symptoms include restlessness, anxiety, dizziness, drowsiness, tremor, ataxia, depression, confusion and coma. Death may occur from depression of the respiratory

or cardiovascular system.

The enzyme inhibition brought about by these compounds, some of which act directly and others only after metabolism, is not readily reversible, and the natural regeneration of cholinesterase is a slow process, the speed being dependent on the compound involved. Repeated exposure may therefore have a cumulative effect though the organophosphorus insecticides are, in contrast to the chlorinated insecticides, rapidly metabolised and excreted and are not appreciably stored in body tissues. 'Ageing' of the cholinesterase may inhibit spontaneous regeneration and render the patient unresponsive to oximes.

There is among this group of insecticides appreciable variation in the severity, latency and persistence of toxic effects.

Malathion (the most frequently used organophosphorus insecticide) - This is one of the least toxic organophosphorus insecticide, the estimated mammalian lethal dose being 1 g. per Kg. body weight. Daily application of 10 per cent malathion dust to the human skin for several weeks has produced slight irritation but little or no inhibition of blood-cholinesterase activity. Maximum permissible atmospheric concentration 10 mg per cubic metre.

Like always a conflicting report - In a modified 'repeated insult' patch test, 25 per cent 'technical' malathion was found to produce extreme sensitisation of the skin. (A.M. Kligman, J Invest Derm, 1966, 47, 393).

#### Toxic effects of Carbamate Insecticides

The carbamates are cholinesterase inhibitors, differing from organophosphorus insecticides in that the inhibition they produce is generally more rapidly reversible. Symptoms of poisoning develop rapidly thus minimising the likelihood of prolonged exposure. Routine estimation of serum cholinesterase activity, as a check on poisoning are of little value.

Understandably there is an overlap in the toxic manifestations of organophosphorus and carbamate insecticides.

#### Toxic effects of Dinitrophenol and related Insecticides

These substances may be absorbed by inhalation, by accidental ingestion, by absorption through the skin. They are cumulative poisons, causing an increase in the metabolic rate, which may lead to death in a manner resembling heat stroke.

Yellow discolouration of the sclera, skin and hair indicates absorption of significant quantities and may precede onset of symptoms. Mild poisoning is usually accompanied by copious sweating and thirst; nevertheless, patients often feel well, owing to the increased metabolic rate.

More severe poisoning is shown by profuse and continuous sweating, fatigue, nausea, abdominal pain, restlessness and loss of weight. Late symptoms include an increase in the rate and depth of respiration, tachycardia and a raised temperature. Death may occur from respiratory and circulatory failure.

Maximum permissible atmospheric concentration of dinitrocresol 200 µg per cubic metre.

The toxic and lethal concentrations in the air of DNOC and related compounds were estimated to be respectively 36 and 100 µg per litre. (E.N. Burkatskaya, Gig. Sanit, 1965, 30, 34 Bull. Hyg. Lond. 1965, 40, 599).

#### Determination of Toxicity Index

The toxic manifestations of the different groups of insecticides have already been mentioned earlier. It may now be worthwhile to dwell a little bit on the toxicity tests, LD<sub>50</sub> and how the acceptable daily intake levels are arrived at.

Tests on the acute toxicity are carried out on several animal species in single and multiple doses to give an

idea of how much chemical it might take to injure or kill exposed humans. In these tests the chemicals are administered by several routes including the mouth, the skin and if necessary, by inhalation. The figure called LD<sub>50</sub> which is calculated represents the dose which kills 50 per cent of the groups of animals and is valuable for comparative purposes. The cause of death is also investigated in order to know whether the lethal dose is relevant to man. For example, a substance that kills by inhibiting an important enzyme is much more likely to be a hazard to man than a substance which kills animals by causing gastro-intestinal irritation when high concentrations are administered. A multiple dose test gives an indication of what is likely to happen to man following multiple exposures over a short period, such as might occur in agricultural practice. Particular attention is paid to the detection of substances which might adversely affect the skin and eyes.

Short-term studies, in which animals receive daily doses of a chemical for about one-tenth of their life span are carried out to determine the possible effects of longer term exposure. These tests are the equivalent of several years exposure in humans. Rats, mice and other species of animals are fed or injected with the chemical daily for several months and they are looked after and examined exactly as are hospital patients. They are observed carefully several times a day, weighed periodically, their food intake is measured and biochemical and haematological tests done to assess their state of health. The only difference from patients is that during the test they are killed by anaesthetic and post-mortem examinations made of all their organs and tissues. Sometimes the tests are modified so that animals which are adversely affected are allowed to recover or are given treatments for poisoning in order to determine the way to treat humans accidentally over-exposed to the chemical. Long term tests are then performed in many ways. These are to study the effects of substances that might accumulate or produce their effects slowly over an even longer period. The most important toxic effect investigated is the carcinogenic potential. Rats and mice are the most convenient species to use and they receive treatment for most of their life-span - this amounts to years in the case of the rat.

Having decided in qualitative terms the ill-effects to be expected in human subjects who might be exposed to a chemical, it is necessary to consider quantitative aspects. This is particularly important where long-term ingestion of small residues in food is involved.

Table 1 - Some Insecticides - their LD<sub>50</sub> and maximum permissible Atmospheric Concentration (MPAC)

Compound	LD <sub>50</sub> (mg/kg body weight)	MPAC mg/m <sup>3</sup>
<b>CARBAMATE</b>		
Carbaryl	400	5
<b>ORGANOPHOSPHORUS</b>		
Dichlorvos	25	1
Malathion	1400	10
Metriphonate	-	Very toxic
Parathion	5	-
<b>ORGANOCHLORIDE</b>		
Dicophane (DDT)	250	1
Dieldrin	-	0.25
BHC	125	0.50
<b>OTHER INSECTICIDES</b>		
Perqual	250	0.50

In general, humans tend to be more sensitive than animals to many chemicals. In addition, any two people do not necessarily react in the same way to a given amount of chemical; a dose which is harmless to one person may make another very sick indeed. When it comes to transferring quantitative animal data to humans, it is necessary to take these factors to account.

In practice, it has been found that the dose of chemical shown to be harmless to animals when given over a long period (for example two years in the rat) is

divided by a safety factor of about 100. This is the maximum acceptable daily intake level.

### Pesticide Plant Worker & his Health Hazards

It is obvious that the workers in a pesticide manufacturing factory would be exposed to pesticides. Their exposure to pesticides is a shade different from that in case of agricultural workers. In some measure this is related to the degree and extent of mechanised and sophisticated handling of the materials. However, pesticide workers are also exposed to other chemical hazards, due to intermediary chemical compounds used and formed in the manufacture of pesticides. These are, more often than not, both toxic and corrosive in nature. Of course, it would be impossible to deal here with all of them. Nevertheless in order to illustrate the point in respect of toxic effects and first aid, two chemicals viz., phosgene and phosphorus pentachloride, which are used in manufacture of pesticides like organophosphorus and organochlorine compounds, is considered here. (For details on hazards of other chemicals the reader is referred to "Hazards in the Chemical Laboratory" ... ed G.D. M. The Royal Institute of Chemistry, London 1971).

#### Phosgene (carbonyl chloride and solutions)

The gas is colourless and has a musty smell. A pale yellow liquid it boils at 7.6°C. Solution of gas in toluene (HIGHLY INFLAMMABLE) is available commercially. **EXTREMELY HARMFUL GAS.**

Do not breathe gas. Avoid contact of solution with skin and eyes. TLV 0.1 ppm (0.4 mg/m<sup>3</sup>).

**Toxic effects** - The gas produces delayed secretion of fluid into the lung (pulmonary oedema) when inhaled and there may be delay of several hours before effects develop. These include breathlessness, cyanosis and coughing up of frothy fluid.

#### First aid -

Vapour inhaled : remove from exposure, rest and keep warm. If inhalation has been considerable medical observation is essential.

Affected eyes : Irrigate thoroughly with water when splashing by a solution has occurred; obtain medical attention.

Skin contact with solution : wash thoroughly with soap and water; remove contaminated clothing.

If solution swallowed : Induce vomiting and afterwards wash out mouth thoroughly with water; obtain medical attention.

#### Spillage disposal -

(a) Solution in toluene: Shut off all possible sources of ignition. Instruct others to keep at safe distance. Wear breathing apparatus and gloves. Apply non-inflammable dispersing agents and work on emulsion with water - run this waste, diluting greatly with running water. If dispersant is not available, absorb on sand, then pour into bucket(s) and transport to safe, open area for burial. Site of spillage should be washed thoroughly with water and soap or detergent. Ventilate area of spill well to dispel remaining vapour.

(b) Gas: Surplus gas or leaking cylinder can be vented slowly into a water-fed scrubbing tower or can be absorbed in a fume cupboard, or into a fume cupboard situated by such a tower.

#### Phosphorus pentachloride

White to pale yellow, fuming, crystalline mass; pungent, unpleasant odour. Violently decomposes water with formation of hydrochloric acid and phosphorus. **IRRITANT VAPOUR AND DUST. CORROSIVE. CAUSES BURNS**

Avoid breathing vapour and dust. Prevent contact with eyes and skin. TLV 1 mg/m<sup>3</sup>.

**Toxic effects** - Vapour and dust severely irritate mucous membranes and all parts of the respiratory system. The vapour and solid burn the skin. If taken into the mouth there would be severe irritation and damage.

**Chronic effects** - Continued exposure to low concentrations of vapour may cause damage to lungs.

## First aid -

Vapour inhaled : remove from exposure, rest and keep warm in severe cases, obtain medical attention.

Affected eyes : irrigate thoroughly with water; in severe cases obtain medical attention.

Skin contact : Drench with water and treat with magnesia/glycerol paste; remove and wash contaminated clothing before reuse; burns must receive medical attention.

If swallowed : wash out mouth thoroughly with water and give plenty of water to drink followed by milk of magnesia; obtain medical attention.

## Spillage disposal -

Wear goggles and gloves. Mix with dry sand, shovel into enamel or polythene bucket, transport to a safe, open area and add, a little at a time, to a large volume of water; diluting greatly with running water. Dispose of sand as normal refuse. Wash spillage site with water.

In 1977-78, 5.8 per cent, i.e. over 4.1 lakh people of the 70,93,000 persons employed in about 85,000 factories, belonged to the chemicals and chemical products group. (data from *Statistical Outline of India, 1982*). The figure by now must have vastly increased. A significant portion of these may be expected to be vulnerable to the potential hazards of toxic and corrosive chemicals.

Some relevant questions that come to my mind are:

1. Are there adequate safety measures instilled in the work environment of these workers?
2. Are the workers adequately informed, trained and educated?
3. What is the level of monitoring and precautionary measures?
4. Is there any sort of medical and health audit?
5. Factory inspections: How effective? How credible?

The spate of gas leaks from various centres leaves behind a dreadful and dismal picture. How should we react to all this effectively? The chemical and intermediary compound hazards, at least the potential hazards, are indeed great. But the potential hazards from the finished pesticides are also no less. The reference quoted below will amply illustrate this.

Liver disease in 8 chemical workers was attributed to contact with dicophane and/or gamma benzene hexachloride (BHC) in the course of their work; none had a history of hepatitis, diabetes, or alcoholism; in 4 patients the disease progressed to complete cirrhosis. The diet of patient with any liver disorder should be free from pesticide residue. (W.Schultman, *Arch-Gewerbehyg*, 1968, **24**, 193 per Abstr. *Hyg.* 1969, **44**, 260).

## Pesticides in Agriculture: Hazards to Workers' Health

Fumigation, spraying, dusting, other modes of application including bait laying of pesticides are the number of ways in which these chemicals assault the health of worker in agriculture. Illiteracy, ignorance and a poor sense of hygiene and sanitation make them even more vulnerable. The following excerpts from Amita Baviskar's paper "Pesticides or Biocides? The Pesticides Problem in India" (available with C.E.D. File K-33 a) brings out these issues more forcefully.

Pesticide applicators and workers in pesticide factories constitute a 'high risk' group as they routinely come into contact with these chemicals. Workers at the Hindustan Insecticide factory in Delhi had a DDT residue, DDA in their urine which was three times higher than that of the general population. Studies carried out in Gujarat on people spraying DDT (1971), BHC (1974) and Malathion (1975) showed the following effects: Though DDT residue levels in serum were 7 times higher in the applicators than in the controls, no long-term effects were noticed except for slightly lowered neurological responses. While BHC residue levels were 3 to 5 times higher in the applicators, no specific clinical symptom was observed, but dermatitis and lack of balance and co-ordination were seen in a few cases. Malathion as an emulsion caused development of toxic symptoms like tremors and even a few fatalities.

As the National Commission on Agriculture has noted, few pesticide sprayers in India, usually illiterate labourers, either in agriculture or in the public health sector use gloves or masks. A study of farm workers engaged in spraying in Gujarat showed that they were not provided with face masks; only 50% covered their nose and mouth with a cloth; and 20% failed to wash after spraying. Dr. Reddy, superintendent and paediatrician at the Niloufer Children's Hospital in Hyderabad describes the conditions under which pesticides are stored and used in India: 'Poor people live in one room only, so they keep the pesticides in the room as well. They live there, cook there and eat there. The powder is in the air. When they spray crops, the spray sometimes drift into the house and children are exposed'. Labelling is also often faulty. A report from Tamilnadu points out that a company has been selling pesticides there whose containers give safety instructions only in English and Hindi whereas the local language is Tamil. Apart from such callousness, illiteracy and poverty are the main cause for not being able to follow directions for the safe application of pesticides. In any case, the standard instruction -- "In case of accident, call physician" -- is often a cruel joke in remote rural areas. Besides, a large amount of pesticides sold in the market are spurious. And now, aerial spraying has been introduced in some states, leading to greater risk of exposure. The blame for direct pesticide poisoning can be thus put partly on the easy availability of toxic pesticides, often with little effective restriction or control, IN CONDITIONS WHERE THE NECESSARY SAFETY PRECAUTIONS ARE HIGHLY UNREALISTIC. Dr. Applestone of WHO has said, "In all societies, but especially where literacy is low, probably the most effective single measure to promote safety is the restriction of the availability of the more toxic pesticides to those who have been trained in their use, and who have a specific need for them!"

As if this is not enough, the situation becomes further alarming when one realizes that many of the agriculture workers are adversely pre-disposed to the myriad of toxic manifestations of chemical pesticides (detailed earlier) which include effects on respiratory and gastrointestinal systems as also the skin and liver. A sizeable number of them are at the start, cases of respiratory (TB/asthma) and liver disorders (alcoholism) and thus extremely vulnerable.

The agricultural workers do not engage themselves with 'pesticide operation' every day but in cities like Bombay, workers engaged in 'pest control services' are exposed to very high concentrations throughout the working hours, day in and day out. One does not normally see them using masks or protective gears. There's a small population (my guess is that it could be 1,000 in the city of Bombay), but their plight merits consideration. It seems very unlikely that their biochemical and physiological parameters are ever monitored and yet none will disagree that these are essential.

## What can be done to remedy the situation?

Polyneuritis developed in 2 men engaged in servicing aircraft used for spraying DNOC on locust swarms in Kenya and Tanganyika. Symptoms cleared completely on removal from exposure (H. Stoll, *Br. Med. J.* 1956, 900).

## Pesticide Residues and the General Population

This particular topic is so clearly dealt with in Amita Baviskar's paper (referred to above) it is felt that it is worthy of reproduction here once more.

There are the hazards of pesticides residues in the harvested crops and in the environment. Residues or "the chemicals present after the initial deposit is established", contribute significantly towards contamination of food, water, air and soil. When present in amounts above safety levels, they can often result in degenerative changes in humans and their environment. While surveys of these changes have been scattered and limited so far, even this scarce data shows alarming trends. It has been found that Indians ingest more pesticides through their food than any other nation studied.

University, National Institute of Occupational Health, Central Food Laboratory and other such bodies have revealed the presence of pesticides residues in levels exceeding the ADI (acceptable daily intake) in all food products. For instance, a 7-year study of foodstuffs from Hyderabad's market showed that 56 per cent had residues of DDT and BHC, 36.6 per cent of these in excess of the prescribed limits. Similar results have been shown elsewhere. This had happened due to indiscriminate pesticide application during the storage of grain and on cattle for parasite control; the use of contaminated water and crop by-products as cattlefeed, improper washing of crops etc. While pesticide residues in milk products are among the highest for all foodstuffs, no maximum residue limits have been fixed for pesticides in animal feed and fodder. The contamination of wheat with parathion led to about 100 deaths in Kerala in 1958. In another instance of wheat poisoned with BHC, some 250 villagers of Uttar Pradesh became victims of epilepsy in 1977-78. It is difficult to remove pesticide residues from food. Boiling may only remove 35% to 60% of organophosphate residue and 20% to 25% of organochlorines. Residues above tolerance limits have been found even in cooked foods. The effects of this invisible insidious poisoning are frightening.

Organochlorine pesticides have the distinction of being the most persistent in the environment - they collect and progressively increase in the tissues of insects, fish, birds and animals. The level of DDT in the body fat of residents of Delhi has been found to be 26 parts per million (ppm) on an average - well above the maximum residue limit of 1.25 ppm - the highest in the world.

DDT in the blood of Indians has been found to be 10 times more than in most other countries. A study shows that DDT and BHC residues in human milk are so high that babies ingest 21 times the ADI recommended by WHO. Another factor affecting the hazard from residues in India is that these residues are far more dangerous when combined with a low-protein diet as in the case with the majority of our population.

#### Legislation in respect of Pesticides in Agriculture (.. & Food)

When one reads about the indiscriminate and rampant use (or misuse) of pesticides, one wonders whether there is any legislative support to fall back on. Indeed there is! (see Paper No. 8). The Insecticides Act 1968 is comprehensive but is ineffective in absence of an enforcing agency. Besides, somewhere in the text of the Act an exemption is mentioned which reads as follows:

38 Exemption - (1) Nothing in this Act shall apply to :

(a) the use of any pesticide by any person for his own household or for his kitchen garden or in respect of any land under his cultivation;

(b) any substance specified or included in the schedule or any preparation containing any one or more such substances, if such substance or preparation is intended for the purpose other than preventing, destroying, repelling or mitigating any insects, rodents, fungi, weeds and other forms of plant or animal life not useful to human beings.

One gets apprehensive whether this could provide a loophole for the offenders. Likewise there is the Prevention of Food Adulteration Act of 1954. It lists the tolerance limits of some 11 insecticides and pesticides. But then again the enforcing agency seems to be lacking. Should this not be the responsibility of FDA? Can we initiate action?

#### Integrated Pest Management (IPM)

With all the hazards of pesticides, the natural question which arises is - Is there no alternative to the use of pesticide? Luckily the answer is in the affirmative and it exists in what is known as Integrated Pest Management (IPM). IPM is an integration of agricultural practices like crop-rotation, tillage, intercropping and sanitation and the environmental and biological control designed to the needs of the specific region. It harnesses the predators to control a wide range of insects and pests.

And happily there are success stories existing well.

Friends Rural Centre, Rasulia, Hosangabad 451 001, M.P. reported that by progressively reducing and ultimately eliminating inputs of fertiliser and pesticides, they were able to increase or atleast maintain the net income from their experimental farm. (Hindu, 24.9.85).

Table 2 - Health and Environmental hazard associated with Pesticides

Pesticide	Associated Hazard
Aldrin/Dieldrin	Carcinogenicity; environmentally persistent; highly toxic if swallowed or inhaled; toxic to fish, birds and bees.
BHC	Oncogenicity.
Carbaryl	Oncogenicity, mutagenicity, teratogenicity; highly toxic to Honeybees, toxicity for humans increases with a low protein diet.
Carbofuran	Acute inhalation and oral toxicity; extremely toxic to birds, fish, shrimp, crab and other wild life; fatal if swallowed.
Chlordane	Oncogenicity; reduction of non-target species; environmentally persistent; fatal if swallowed.
DDT	Virtually nondegradable; suspected carcinogen
Diazinon	Contact poison, fatal to shrimp & crab.
Parathion	Extremely high, acute inhalation & dermal toxicity.
Phosphamidon	Acute dermal toxicity
Toxaphene	Tumour induction, environmentally persistent.

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The use of chemicals to kill pests is an ago-old practice. Before the advent of synthetic organic pesticides around 1940, simple compounds -- mainly arsenicals -- were used to kill pests. One such compound called Paris Green (copper aceto-arsenite) is applied even today to pools and stagnant water in India, to control malaria-transmitting mosquitoes. Health officials maintain that such waters are safe even for drinking since the level of Paris Green used is well within the safe limits. However, arsenical pesticides have been found to be extremely persistent. They can remain in soils for upto 40 years. No information is available from India on the residues of arsenical pesticides.

Synthetic pesticides fall into three categories viz. organochlorine, organophosphorus and carbamat compounds. Additionally, there are pesticides of natural origin such as nicotine, pyrethrin and rotenone. Of these, organochlorine compounds such as DDT and BHC are the ones most widely used in developing countries. The current annual consumption of DDT is nearly 12,000 tonnes for public health & 2,000 tonnes for agriculture (*Indian Express*, Sept. 28, 1985). The projected demand for DDT & BHC during 1984-85 was 17,750 and 42,500 tonnes (MT). This is expected to increase to 19,750 and 47,000 MT by 1989-90 (*Pesticide Information*, July-Sept., 1984).

While a majority of the pesticides do not persist in soil or water for more than a few weeks or at the most months, the few that do include organochlorine insecticides. While the spraying of these compounds may reduce the need for frequent applications, it increases the ecological risks.

The realisation and fear of the possibility of the adverse ecological impact of pesticides began in 1950. The book *Silent Spring* by Rachel Carlson, published around 1966, focussed attention to the problem in an exaggerated but effective way.

In India, the Bengal Famine of 1942 rudely focussed attention on to agricultural practices. A report published subsequently outlined the factors that led to the food shortage and jolted agriculturists into awareness about the advantages of using pesticides to save precious foodgrains from destruction. However, pesticide use began only in 1950s. India's present population of 630 million may go beyond 1,000 million by the turn of the century. By then the demand for foodgrains will be 235 million as against the present production of 135 million. Today, 25-30 per cent of our foodgrain is destroyed by pests. In the face of these statistics, it is obvious that the use of pesticides will have to continue. Also pesticides will have to be used to control vector born diseases such as malaria and filariasis. However, even the champions of pesticides are now cautioning against excessive and careless use and recommend that organochlorine pesticides be phased out gradually. Though the use of pesticides in India is far below that of most developed countries, pesticides residues in Indian foods, and in the bodies of the Indian people are higher than those reported from the developed countries. It is therefore clear that judicious and proper use can reduce the environmental hazards of pesticides considerably. In many developing countries, the use of organochlorine compounds has come down markedly. Less persistent but expensive, organophosphates and carbamates have gained importance. The type of pesticides used will ofcourse vary depending upon the nature of pests to be combated.

In 1967, the government of India constituted an expert committee to examine the pesticides used and its hazards. According to the committee's report, most fatal accidents are due to carelessness and ignorance. The committee recommended stricter control on manufacture, sale and use of pesticides. The result was the Insecticide Act of 1971. The committee also proposed setting up of laboratories to monitor pesticide residues in water and food. A few laboratories have now been equipped for the purpose. They include The Central Plant Protection and Training Institute (Hyderabad), Indian Agricultural Training Institute (Delhi), The Central Food Technological Research Institute (Mysore), Central Food Laboratory (Calcutta), Punjab Agricultural University (Ludhiana) and

## PESTICIDE RESIDUES IN FOOD AND IN HUMANS

Dr. Mahtab S. Bamji

**Table 1**

**PESTICIDE USAGE PATTERN IN INDIA**  
(Metric tonnes)

	1973-74	1983-84
Chlorinates	32,625	65,085
Phosphates	3,570	23,000
Carbamates	2,510	6,310
Plant based compounds	50	120
Fumigants	1,150	1,325
Fungicides	10,337	12,550
Weedicides	810	2,770
Rodenticides	550	850
Ascarcides	130	220
Nematocides	25	55
Plant growth regulators	15	55
<b>Total</b>	<b>51,772</b>	<b>1,12,340</b>

Source : Krishnamurthy, 1985

**Table 2**

**DDT CONTENT IN SOME FOOD**  
(ppm)

Wheat	1.6 - 76.0
Rice	1.0 - 16.0
Pulses	3.0 - 35.0
Groundnut	3.0 - 35.0
Vegetables	0.5 - 05.0
Potatoes	68.5

Source : Krishnamurthy, 1985

**Pesticide Residues in Foods**

The data generated so far shows wide variation in values for organochlorine pesticide residues in food items. However a substantial proportion of food items have been found to have pesticide load in excess of the limits permitted by FAO/WHO (Table 2) (Krishnamurthy, 1985). The reported mean values for daily intakes of DDT & its metabolites has varied between 100-200 µg per day (Gupta et al, 1984, Kaphalia et al, 1985). Non-vegetarian diets tend to contain higher amounts. These figures work out to values close to or above 5 µg/kg body weight per day regarded as Acceptable Daily Intake (ADI) by FAO/WHO (WHO, 1970). The corresponding figures for daily intake of DDT/kg body weight from the USA were 0.9 µg in 1965 and 0.4 µg in 1970; and in the U.K. it was 0.8 µg in 1965 and 0.2 µg in 1970. The downward trend in these countries were achieved by banning or restricting the use of organochlorine compounds and by educating about safe usage. A recent report of the Global Environmental Monitoring System says that the levels of DDT and Gamma-Xane residues in human breast-milk in India and China are so high that an average infant would consume 3-4 times the admissible daily intake permitted by FAO/WHO (Krishnamurthy, 1985).

**Pesticide Residues in Humans**

Pesticides can enter human systems through direct exposure — occupational (agricultural labourers, public health workers involved in spraying operations and workers in factories manufacturing pesticides), accidental, experimental or through ingestion of contaminated food and water — surface contaminated and food chain. Animals, birds and fish can concentrate pesticides in body fat and pass the load to humans through food such as meat, fish, milk and eggs.

The human pesticide residue is a biological index of pesticide exposure. In acute intoxication, the residue provides diagnostic information; in the occupationally exposed, the residue is a surveillance tool reflecting industrial exposure; in the general population, the residue is a measure of incidental exposure. Average levels of the persistent pesticides in fat and blood have been used to express the level of pollution of the population at large with these chemicals. (Davies, 1973).

NIOH, Ahmedabad has examined the levels of DDT in adipose tissue of autopsy samples from Calcutta and Bangalore (Gupta et al, 1980, 1982). The mean levels of DDT and BHC in Calcutta samples were 6.25 ppm (parts per million) and 1.62 ppm. Males had higher concentrations of residues than females, but this may be because of larger adiposity in females. The total body burdens may not be different. Older persons had higher levels than younger persons. Similar values were observed in Bangalore by the same workers and in Delhi by Bhaskaran et al (1979). BHC levels tended to be higher in Bangalore. Earlier reports mention much higher levels of DDT and BHC in the body fat of Indians. (Dale et al 1965). Whether this difference is real or methodological is not clear. Serum of food animals, chicken and humans in Lucknow area had a higher concentration of DDE (a metabolite of DDT) than DDT (Kaphalia and Seth, 1984). This indicates cumulative or chronic rather than acute exposure. Total DDT levels in human serum were higher than in other species, suggesting biomagnification. However, the levels of DDT and BHC were well within the acceptable limits.

In recent years, attempts have been made to study adipose tissue levels in relation to disease. Some reports have indicated higher levels of DDT and its metabolites in a variety of pathological conditions including cancer, hypertension and diseases of the liver. Higher blood and placental tissue levels have been reported in pre-term rather than full-term babies (Saxena et al, 1980). Whether the higher levels are the cause or the effect of the conditions mentioned is a moot point. Metabolism of pesticides and other foreign chemicals by the human body can be modified by previous exposure and consequent induction of microsomal drug metabolising enzymes. Use of drugs is also known to modify the body's capacity

to handle pesticides. By and large the significance of body burden of pesticides in terms of human health is not known. Health profile of workers occupationally exposed to DDT, Aldrin, Deeldrin, Telodrin and Endrin has not revealed any disturbing trends. Effects of malnutrition on the tolerance of pesticide by the body needs to be examined. Animal studies suggest that protein deficiency increases though marginally, acute as well as chronic toxicity of several pesticides (Boyd, 1972).

Most toxicity studies are conducted in animals and LD<sub>50</sub> is taken as a measure of safety. Several pesticides have been found to produce cancer, congenital malformations, liver and kidney damage, vomiting, neurological problems, eye damage and sterility. Very often the company manufacturing the pesticide does not reveal the data on acute and chronic toxicity. The Bhopal accident emphasises the need to know, not only the short and long term effects of the final product, but also the intermediates which may be stored in a factory. This applies to all hazardous chemicals. The case of the Handigodu syndrome that hundreds of villagers of Shimoga (Karnataka) suffered from, between 1969 and 1977 is well known by now. It was only in 1975 that epidemiologists discovered that this disease was a result of consuming crab and fish contaminated with endrine and DDT. (see Paper No. 1 for details).

Nothing sums up the argument against careless, excessive and irrational use of pesticides better than these figures reported by the WHO and UNEP: Every year 4,00,000 people in the Third World are poisoned by pesticides and 22,000 of them suffer death. It is also relevant that most of these pesticides are banned in the West but are nonetheless manufactured and marketed by multi-national companies in Third World countries like India.

Table 3 - Pesticides used in India and banned or restricted abroad

Pesticide	Some countries in which it is banned or restricted
Aldrin	USA, UK, W. Germany, Sweden, Canada, Italy, USSR, New Zealand.
BHC	USA, Japan, USSR, Denmark, Sweden, France, W. Germany
Chlordane	Argentina, USA, UK, Turkey, Sweden, Denmark, Bulgaria, Italy.
DDT	Australia, UK, USA, Colombia, Greece, USSR, Poland, Switzerland
Endrin	Argentina, UK, USA, Japan, Mexico, W. Germany, Finland.
Parathion	Italy, Japan, Spain.
Toxaphene	Argentina, Finland, W. Germany, Italy.

Source : Amita Baviskar's Paper

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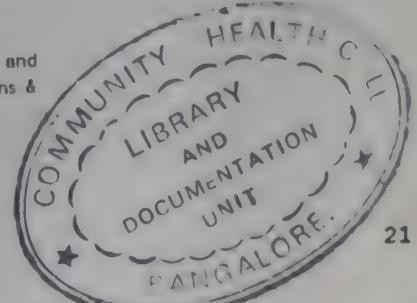
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PESTICIDES MANUFACTURED IN INDIA (1983)  
**PRODUCTION CAPACITIES OF TECHNICAL GRADE  
 PESTICIDES**  
 (As on 1st October, 1983)

	Licensed	Installed	Licensed	Installed
<b>A. INSECTICIDES</b>				
BHC	41,900	41,900	Ferbam	
Carbaryl	7,000	7,000	Metham Sodium	
DDT	9,088	9,144	Carbondazim (M.B.C.)	635 335
Methyl Demeton (Metasystox)			Sulphur	2,000
Phorate	1,050	1,050	Carboxin	-
Monocrotophos	950	950	Calixin	
Tetradifon	18	18	Kitazino	
Ethion	200	200	Dodine	
Diazinon	250	-	Guazatine	
Temephos			Captafol	
Pyrethrum Extract			Captan	13,084 13,01
Fenthion	-	-	<b>C. RODENTICIDES</b>	
Carbofuran			Coumafuryl	
Chlorpyriphos			Zinc Phosphide	1,294 1,29
Methamidophos			Warfarin	50 5
Orthene	-	-	<b>D. HERBICIDES AND PLANT GROWTH      REGULANTS</b>	
Synthetic Pyrethroids			2,4-D	2,835 2,0
Fenvalerate	100	100	Dalapon	250 2
Cypermethrin	-	-	Paraquat	
Cypermethrin			Propanil/Nitrofen	1,500 1,50
Permethrin	-	-	Fluchloralin	
Endosulphan	4,000	4,000	Diuron	200 20
DDVP	376	276	Alpha Neptyl	
Phosalone	1,000	1,000	Acetic acid	20
Lindane			Cycocel	231 1
Dimethoate	2,050	1,550	Butachlor	-
Fenitrothion	1,400	1,100	Isoproturnon	-
Malathion	18,410	11,990	Tribunil	
Methyl Parathion	1,850	1,850	Simazine/Atrazine	-
Phoshamidon	836	836	Glyphosate	-
Quinalphos	800	500	<b>TOTAL HERBICIDES &amp; PGRs</b>	5,736 4,
Phenthroate	600	600		
Nicotine Sulphate	40	40		
Decamethrin	93,689	84,184		
<b>B. FUNGICIDES</b>				
Copper Oxychloride	3,420	3,660	<b>E. FUMIGANTS</b>	
Nickel chloride			Aluminium	
Copper Sulphate			Phosphide	1,820 1,
Organo Mercurials	95	91	E.D.B.	258
Zincb Maneb	2,500	2,500	M.B.	300
Thiram	400	400		
Ziram	634	634		

The ways in which pesticides produce their toxic effects in the body are extremely important because the pesticides have become substances of common use in every aspect of life. However, not many long term human studies are available which pin point the various biochemical and molecular disturbances caused by pesticides. This lacunae in the knowledge of pesticides limits the possibility of properly defining the pathology, understanding the mechanisms of actions of pesticides and developing antidotes against them. However, it is felt that available information could be reviewed so as to develop proper understanding about toxicodynamics for the benefit of interested workers in this field.

### General Considerations

#### Definition of a poison

A poison is a substance of exogenous origin that in excess or in small quantity causes illness/death by chemical interaction with protoplasm.

#### Poisoning

The poison may damage locally as a corrosive or an irritant, produce systemic injury after absorption or combine both the effects. Poisoning is a sum total of all the primary and secondary effects of a poison. The state of poisoning may be simple as with acute carbon monoxide produced anoxia or it may become exceedingly complex as functional and structural systems of the body adapt and interact to the presence of a long-standing noxious chemical stimulus.

#### Biochemical lesions

It implies precise chemical change by which the poison acts harmfully. The ultimate target of the toxic chemical is the cell. Within the cell exists a complex of chemical substances in dynamic equilibrium. When a new substance is introduced into this normal scheme of things adjustments must be made if homeostasis is to continue. Homeostasis is nothing but the narrow range in which the physical and chemical conditions (e.g. temperature, pH concentration of various ions and non-ionic substances of the intracellular fluid which bathes the cell organelles) are maintained.

If the interjected substance is inert or if it is deactivated or removed promptly, the consequent intra-cellular chemical disturbances or energy change may remain within the normal range. The episode terminates and remains sub-clinical. The state of poisoning may be said to have taken place when the substance leads to physicochemical disturbance sufficiently severe to have produced demonstrable change in structure or function. This definition (*Text of Pathology - Anderson*) however seems to be inadequate because the sub-clinical effects at a given time are likely to reduce the biological reserve of the organism to face the chemical disturbances produced by poisons during subsequent exposures. WHO Technical Report Series No. 677, 1983 on pesticide research adopts the following criteria for defining the adverse effects of pesticides:

1. Effects that indicate early stage of clinical disease.
2. Effects that are not readily reversible and indicate a decrease in the body's ability to maintain homeostasis.
3. Effects that enhance the susceptibility of the individual to deleterious effects of other environmental influences.
4. Effects that cause relevant measurements to be outside the normal range, if they are considered as an early indication of decreased functional capacity and
5. Effects that indicate important metabolic and biochemical changes.

The cell converts its aqueous chemical mix into a variety of products for growth, reproduction, secretion,

etc. by enzymatic action and the by-products of metabolism for excretion. The most vulnerable component of all the cells are enzymes. Proteins are peculiarly susceptible (all enzymes are protein by nature) to the denaturing effect of a poison. Inference with cellular oxidation mechanisms also may constitute the common early manifestation of a diverse group of foreign chemicals.

Pathological lesions due to poison: (1) Local injury (2) Systemic injury after absorption (3) Combined local and systemic effects (4) Non-specific effects due to anoxia, secondary shock, renal failure etc. (5) No detectable lesions.

Among primary reasons for localisation is the concentration of the chemical at the portals of entry and excretion. Many poisons (such as corrosive agents, many heavy metals, the war gases etc.) produce injury immediately upon contact with tissue. The lesions are non-specific, produce simple necrosis and are not readily identifiable.

The skin, the gastro-intestinal tract and the respiratory tract constitute the three principle locations in which the initial contact with the chemical occurs. In all these three sites accentuation of injury may occur if the organ also serves as the portal of excretion, e.g. carbon tetrachloride toxicity in the liver.

Some organs appear to be more sensitive than others to effects of a systematically circulating poison although the exact cause may be problematic.

In a number of instances no morphologic changes can be attributed under light microscopy to the direct chemical action of the agent. This is particularly true for poisons that are rapidly fatal. However, with electron microscopy levels of structural alteration antecedent to cellular necrosis can be assessed and in turn correlated with concomitant change in function.

### Modifying factors

Many factors inherent in the chemical or the host may modify the resultant toxic manifestations of exposures. Factors operative in that are age, sex, general health and hypersensitivity make up. The factors inherent in chemicals are also important, e.g. solubility in water and fats. The nature of catabolic product of the chemical may modify the resultant toxic manifestations.

## Diagnosis

### Toxicological

### Isolation of poison in tissues in lethal quantities.

## Pathological

Characteristic morphological lesions caused by the isolated chemicals.

The problem of diagnosis is important from the pathophysiologic point of view as delayed deaths take place after the poison has been excreted or metabolised. If neither the clinical signs and symptoms nor the pathological lesions are pathognomonic, proof of poisoning rests on the knowledge of adequate exposure. This hardly constitutes definitive evidence for medico legal purposes. The result is that many cases of poisoning go unreported either because they are unsuspected or because sufficient proof is lacking. One must also not forget the toxicity of the vehicle, e.g. kerosene.

## The Organophosphorus Insecticides

This class of insecticides are potent cholinesterase enzyme inhibitors. They irreversibly bind with the catalytic site of the enzyme by competing with Ach, which is a neurotransmitter. Cholinesterase is an enzyme which normally destroys acetyl choline. Ach has a parasympathomimetic effect. It is secreted in preganglionic sympathetic nerve terminals, post ganglionic parasympathetic nerve terminals to sweat glands and myoneural junctions. The true cholinesterase destroys only acetyl choline while pseudocholinesterase can act on other substrates also.

The various effects caused by parasympathetic stimulation are similar to the effects of organophosphorus poisoning because of the accumulation of the parasympathetic neurotransmitter acetyl choline.

#### The Effects of Parasympathetic Stimulation

Effective Organs	Responses
<u>Eye</u>	
Radial muscle,	Contraction (miosis) ↔↔
sphincter muscle, iris	
Ciliary muscle	Contraction for near vision ↔↔
<u>Heart</u>	
SA Node	Decrease in heart rate vagal arrest ↔↔
Atria	Decrease in contractility and increase in conduction velocity ↔↔
His Parkins system	Little effect
Ventricles	Slight decrease in contractility
<u>Arterioles</u>	
Coronary	Dilatation +
Skin and mucosa	Dilatation +
Cerebral	Dilatation +
Pulmonary	Dilatation +
Abdominal viscera, renal	-
Salivary glands	Dilatation ↔
Veins (Systemic)	-
<u>Lungs</u>	
Bronchial muscle	Contraction ↔
Bronchial glands	Stimulation ↔↔
<u>Stomach</u>	
Motility & tone	Increase ↔↔
Sphincters	Relaxation (usually) +
Secretion	Stimulation ↔
<u>Gall bladder &amp; ducts</u>	Contraction
<u>Urinary bladder</u>	-
Detrusor	Contraction ↔↔
Trigone & sphincter	Relaxation ↔↔
<u>Ureter</u>	
Motility & tone	Increased +
<u>Sex organs, male</u>	Erection, variable
<u>Skin</u>	
Pilomotor muscle	-
Sweat glands	Generalised secretion ↔↔
Spleen capsule	-
Adrenal medulla	Secretion of epinephrine or non-epinephrin

The organophosphorus pesticides can produce stimulation followed by depression or paralysis of all autonomic ganglia and skeletal muscle and stimulation and subsequent depression of cholinoreceptive sites in CNS because persistent depolarisation of cell membrane. These effects as expected are expressed as miosis and spasm of accommodation, hypermobility of gastro-intestinal tract, hypersecretory state of various glands, fibrillation, fasciculation and blockade of myoneural junction, bradycardia and hypotension or depression of various centres in CNS especially, respiratory centres.

## Malathion

In mammals malathion itself has only a slight direct inhibitory effect on cholinesterase and non specific esterases (pseudocholinesterase) but one of its metabolites malaoxon is an active inhibitor of these esterases irreversibly reacts to destroy their action.

Clinical poisoning manifests itself within 5 minutes to 3 hours after ingestion of malathion. It is readily absorbed through the GI tract, respiratory tract and the skin. It is widely distributed in tissues with highest concentration in liver.

The malathion and its biotransformation product malaoxan are rapidly hydrolysed by phosphoryl phosphotriesterase, carboxyl esterase, carboxyuridase. The enzymes not inhibited by organophosphorus compounds and hence in mammals the detoxification is more efficient.

compared to insects in whom there is paucity of these enzymes. However, impurities in commercial preparations do inhibit these enzymes and lead to increased toxicity in mammals also.

Rats exposed to diet providing 240 mg of malathion for five months exhibited a smaller than average litter size and number of youngs alive at seven & twentyone days was about half the number in control group. The weight of the young 9 weeks after birth was lower than that of controls. The teratogenicity of malathion is not yet demonstrated experimentally.

In one of the studies in Pakistan (1976) out of 79 workers 16 showed neurasthenia, high reticulocyte count, increased glutathione content as well as cholinesterase inhibition. In some the intoxication was characterised neurasthenic autonomic-vascular syndrome and what was also recalled as angiodystonic syndrome. These subjects had 35 to 50 per cent inhibition of plasma cholinesterase.

### Carbaryl

This is a carbamate derivative acting by inhibition of cholinesterase enzyme. The liver is the primary site of metabolism. It is hydrolysed or hydro-oxylated by enzyme system called mixed function oxydase or microsomal enzymes.

Teratogenicity of carbaryl is reported in beagle dogs and in guinea pigs; weak mutagenicity is also reported.

CNS is affected in rats exposed to carbaryl (20 mg/kg per day) for fifty days. In male rats reduction of sperm mobility and shorter duration of sperm survival and inhibition of spermatogenesis were observed at doses of 2-5 mg/kg given for three to twelve months from second to fourth generation. Changes also include dystrophic and trophic changes in germinal epithelium. A significant decrease in the number of seminiferous tubules was observed at the dose of 5 mg/kg/day. At higher doses (300 mg/kg/day) sterility is observed. In female rats disruption of estrous cycle was observed at doses of 100 to 300 mg/kg/day. The effect increased from generation to generation. Fertility decreased progressively from 2nd to 4th generation.

In rats and dogs carbaryl given during early phase of gestation produced malformations and reduced number of litters born alive.

Carcinogenic potential has also been reported but not conclusively. There is very little information in the relationship between the concentration of carbaryl in air and biological effects in subjects who are occupationally exposed.

### Lindane & Benzene hexachloride

This is a CNS stimulant if present in large systemic concentration, and produces muscle cramps and typeneflexia tremors in man, myriasis or miosis, slow reactivity of pupils. It is also an inducer of liver enzymes. It is known to cause liver damage including liver tumors in mice. It causes fatty degeneration and enlargement of liver cells and glomemlonephitis and pneumonia also.

In the body lindane is distributed to various organs but is mainly stored in fatty tissues. Moreover, it accumulates in brain. The young are more susceptible to lindane than adults. In children concentration of 20 gm/lit in blood was found to be associated with consumptions and a concentration of 600 gm/lit induced hypertension, acute renal failure, acidemia, anaemia and subsequent pancreatitis. In workers in lindane manufacturing plants it was found that those with blood concentration exceeding 20 mg/lit of blood showed non specific electro encephalographic changes and other clinical symptoms.

### Pyrethroids

These are naturally occurring insecticidal esters obtained from pyrethrum flowers grown mainly in Kenya.

#### Mode of action

Nerve excitation is caused by increased negative after potential and repetitive firing following electrical stimulation of nerve at temperatures above 26°C. while nerve blockade occurs at lower temperature. Voltage clamp studies show that these insecticides shift the sodium activation curve in the direction of depolarisation and the sodium inactivation curve in the direction of hyperpolarization. One or both of these mechanisms could account for repetitive nerve firing and nerve blocking. These insecticides also result in a delay in the closing of activation (m gate) which causes an increase and prolonged sodium tail current. They also prolong the following phase of transient sodium current (n gate) without affecting the rising phase.

### Explanatory notes on Medical terminology

<b>Protoplasm</b>	Viscid, translucent colloid material, the essential constituent of living cells.
<b>Anoxia</b>	Absence or deficiency of oxygen, reduction of oxygen in body tissues below physiologic level.
<b>Pathognomonic</b>	Specifically distinctive or characteristic of a disease or pathologic conditions.
<b>Acetyl choline and cholinesterase</b>	Acetyl choline is a neurotransmitter enzyme. It is broken into choline and acetate by acetyl cholinesterase, present at cholinergic nerve terminal. That is, cholinesterase brings to end the neurotransmitting action of acetyl choline. Organophosphorus compounds irreversibly bind cholinesterase and inhibit its function of hydrolysing acetyl choline. The net effect is that acetyl choline gets accumulated at various neurotransmission sites and produces continuous stimulation of that part of the nervous system.
<b>Parasympathetic effect</b>	Effects which resemble those of stimulation of the parasympathetic nerve supply of a part. The function of parasympathetic nervous system include: narrowing of the pupil, gastro-intestinal secretion and peristalsis with relaxation of intestinal sphincters slowing of the heart and reduction of blood pressure. As all these functions contribute to conservative, restorative vegetative functions they are characterised as <u>tropotrophic</u> functions.
<b>Myoneural junction</b>	Junction between nerve and muscle tissues.
<b>Artria</b>	Upper chambers of the heart.

Ventricles

Lower chambers of the heart.

S.A. Node

Sino-atrial node - A group of specialised cells placed in the back wall of the right atrium. The electrical current starts from here and spreads to the rest of the heart resulting in contraction of the heart.

A.V. Node

Atrio-ventricular node - A group of specialised cells placed in the right atrium below the S.A. Node. Conducts electrical current from S.A. Node to the rest of the heart muscles.

Arterioles

Branches of the arteries of the kidney (going to the medullary pyramids).

Detrusor

An involuntary muscle forming a network on the urinary bladder. Contraction of the detrusor decreases bladder n/volume thereby resulting in urination.

Pilomotor muscles

Small bundles of smooth muscle fibres at the root of the hair. The contraction of this muscles causes the hair to stand on end ("goose flesh").

Adrenal medulla

The inside portion of the adrenal gland (placed above the kidneys) produces hormones like adrenaline and noradrenaline.

Autonomic ganglia

Peripheral nervous mechanisms that control smooth muscles, cardiac muscles and gland are referred to as autonomic nervous system (It has two components, sympathetic and parasympathetic). This system has numerous relays between the central nervous system and the viscera in numerous (autonomic) peripheral ganglia.

Miosis

Excessive contraction of pupils.

Fibrillation

A transitory muscular contraction resulting from spontaneous activation of single muscle fibres supplied by a single motor nerve filament.

Neurasthenia

Excessive fatigue of neurotic origin.

Reticulocyte

A non-nucleated cell of the erythrocytic series (i.e. development stage of erythrocyte - red blood cell), grossly indistinguishable from an erythrocyte, but on special staining shows granules or diffuses network of fibres. The red blood cell develops from it.

Glutathione

A compound in animal and plant tissues which acts as a carrier of oxygen.

Mydriasis

Great dilation of the pupil.

Acidemia

Abnormal acidity of the blood.

- Notes compiled by Amar Jesani and Sanjeev Kulkarni

# DANGERS OF PESTICIDES A HORROR STORY....

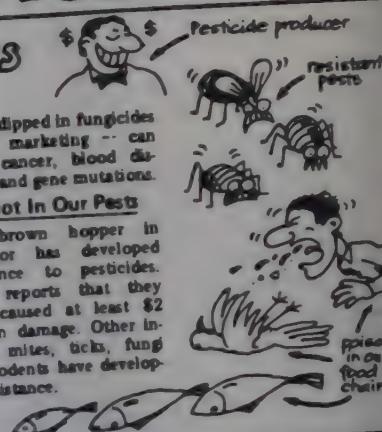
## 1 The Problems

### In Our Bodies

5 times more pesticide elements in the blood of the average Malaysian than in the average American.

### But Not In Our Pests

The brown hopper in Selangor has developed resistance to pesticides. SAM reports that they have caused at least \$2 million damage. Other insects, mites, ticks, fungi and rodents have developed resistance.



### In Our Foods

Benzene Hexachloride in rice, carrots and barley; Epoxyd in cucumbers;

mutate in the body. May cause cancer.

HEPTACHLOR - found to cause liver disease in cases of occupational exposure.

ALDRIN - suspected carcinogen. May cause liver and kidney degeneration. Suspected of causing sterility.



## 2 Dumping

Pesticides and insecticides banned in 20-30 countries, including UK, US, and W. Germany, are still widely used in Malaysia.

DDT - very toxic when dissolved in oil. Can cause cancer and disintegration of liver cells.

DIELDRIN - about 5 times as toxic as DDT if swallowed. 40 times as toxic when absorbed through the skin in solution form. Affects nervous system. May also cause cancer.

CHLORDANE - may cause poisoning if inhaled. Can penetrate skin and accu-

year. But what has it done about it?

One of the pesticides involved include ROUNDUP, a popular weedkiller which continues to be advertised and widely used here.



## 3 The Truth

58 pesticides used in Malaysia were certified safe based on falsified data from the International Bio-Test Laboratory (IBT), the largest commercial lab. in the U.S.A.

The Malaysian Pesticides Board has been aware of the scandal for two

## 4 Infamous Names

### 2,4-D and 2,4,5-T

- Herbicides used in nearly all rubber and oil palm plantations. Mixed together, they make up the notorious AGENT ORANGE, which the Americans used in the Vietnam War.
- Causes miscarriages, birth defects, cancer, where AGENT ORANGE was sprayed in Vietnam, stillbirths rose by 49%.
- Easily available and also sold in sauce bottles.

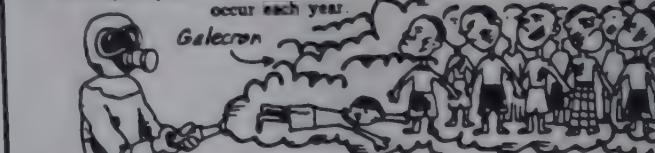
### Paraquat

- A weedkiller commonly used in Malaysia. Popularly known as "KOPI-O" and can be easily bought. Often sold in drink bottles.
- Causes cancer, birth deformities, gene mutation and many other ill effects.
- Used in many suicide cases yet government does not control its sale and use.



## 5 More Evils

- A cancer-causing pesticide, GALECRON, was tested by a Swiss Company on 12 Egyptian children. Someone in the Third World is poisoned by pesticides every minute (W.H.O.)
- At least 6,000 pesticide-related deaths occur each year.



courtesy: CAP Malaysia

The availability and relevance of knowledge in a class society is controlled by the dominant classes and is influenced by their concerns. The production of knowledge is never an objective exercise. The objectives of any study or research programme, stated as well as unstated, determine the methods and materials used which in turn overtly and covertly influence the conclusions reached. In fact the very pattern of available information, i.e. the dearth of information in some areas and the repetitive and exhaustive material in others, is a reflection of the interests of those who control the production and circulation of information. The kind of information that gets accumulated and is generally accessible is such that it does not in any way upset the concerns of the producers (or for whom it is produced). The lack of information is itself used to generate and reinforce certain impressions.

For instance, the dearth of information on MIC was used to create a myth that the gas was hardly worth toxicological notice and therefore had not been researched. The kind of information on it was mostly small population animal studies which could always be challenged. Similarly little was known about dioxin and its toxic effects before the Seveso disaster. In fact it is only since Bhopal that cell death and cell injury in animals and humans are being studied. Apparently books on cells describe "in great detail how cells grow, use energy and divide, but few even mention what happens when cells are injured and how such injury might result in death".

Therefore, in order to do any work in the politically sensitive area of environmental health, we need to know not only how to interpret the paucity of information but also how best to utilise establishment-generated information.

#### Distortion of Information

The degree of hazards in industry is determined by a combination of political and economic factors, with medical factors being mediated by the first two. Safety and production costs are linked such that even the determination of safe limits is itself controlled by the cost of the technology required for implementing it. Moreover the nexus between the owners of industry and the state/government is such that even the nature of safety and health legislation which are passed tend to favour industry rather than people. (Any pro-people legislation is a hard won 'compromise'). More importantly, the interpretation of the nature of extent of damage to health and safety lies with the health professionals who are either employed by industry or government and/or socially, culturally and economically part of the same sections of society as the owners and managers of industry. Thus every recognition and acknowledgement of industry-related ill health or injury is in the nature of a hard won victory.

However, with the increasing awareness of people on environmental issues industry finds itself in a bind. The older 'methods' of safeguarding the interests of industry may no longer be sufficient in the face of the pressure being put on it to introduce safety norms. On the one hand industry is forced to investigate potential and existing hazards and make them known to the people. And on the other, it now has less opportunity to avoid safety practices, processes or other measures for ensuring health and safety. Increasingly, industry and government will seek to influence and manipulate the information being generated about hazards. (One way of doing this is to set up innocuous sounding research institutes which will then undertake 'independent' studies designed to achieve certain objectives.) What are the features/faults/loopholes/biases that one needs to be aware of in these studies?

##### i. Purpose of Study

Every research study has a set of stated objectives and an often unstated purpose. Before attempting to utilise or accept information/conclusions of these studies one needs to be aware of these objectives. Let us look at

three instances to illustrate the point.

In 1965, a 15-year study was initiated at the Hanford Atomic Plant in the U.S. to determine the effects of low-level radiation on workers.<sup>2</sup> Although this was the stated objective, the study was in fact meant only to prove to employees that the exposure levels were quite safe. This became obvious only when an independent researcher noticed an increase in cancers among the employees and notified the authorities. Instead of taking this into account in the study and investigating further, the authorities chose to abruptly transfer the control of the study from an independent university to the employer. The scientists involved in the study were quite aware that the unstated objectives of the study had so influenced the design that no increase in cancers would possibly be detected in the study. Thus if it had not been for the incidental observations on cancers by an independent observer, the study would have proved that the employees had no cause for concern.

Closer home, the ICMR studies on Bhopal must also be examined for their objectives. Most of these studies or atleast the earliest of them were directed at studying the impact of the disaster on particular organs or systems in the human body. At first the objectives seem innocuous enough but when you consider that these studies were being conducted in the absence of any epidemiological data, then they become questionable. By defining these medical studies in terms of organs and systems, the ICMR was leaving out the politically sensitive question of how the gas/gases affected the 'whole' human body and mind. In other words the objectives were such that they incorporated certain loopholes; since the human system as a whole was 'missing' from the scope of these studies, there would always be an indeterminate area of say, the consequences of interacting physiological functions which would form an escape clause for 'sensitive' conclusions.

A third illustration is the occupational studies conducted by the Central Labour Institute at the staple fibre plant of Gwalior Rayons at Nagda. As is well known, the study was a 'response' to an earlier independent study published by the People's Union of Civil Liberties which showed clearly the health impact of the work within the factory. Thus the unstated political objective was to refute the findings of the PUCL publication. Signs and symptoms noted were dismissed as being unworthy of further study and there was deliberate omission of certain aspects<sup>3</sup> which might have revealed 'uncomfortable' information.

Therefore, no matter what the stated objectives of the industry or government study, they must be placed within the context of existing socio-political climate e.g. the level of awareness of the population, the status of working class movement etc. and the level of background information already available.

## ii. Methods and Materials of Study

The design of medico-social study is determined by the objectives of the study. Obviously, then if the objectives are biased, the methods, design and materials would also reveal the biases. (This no doubt raises the more fundamental question of the objectivity and neutrality of scientific method which is beyond the scope of this note.) Moreover, inadequate or faulty design may be deliberately adopted.

Again some illustrations. In Hanford study for instance the number of workers at the plant was considered by experts to be too small to detect the smaller effects of low-level radiation, if they existed. Although this small sample conflicted with the norms of 'good' (what was considered 'good') design the Atomic Energy Commission went ahead and funded the study. The supposedly inadequate design would have served another purpose -- in the event that the study did reveal radiation effects, it could always be explained away on the grounds that the design was faulty! As things worked out, the presuppositions of the Hanford study, that there were no radiation effects and the design sample was too small, were both proved incorrect. (The increase in cancers among the employees came to light outside the study, quite accidentally, in the course of a research study surveying morbidity by occupation in the area.)

The materials and tools used in the study also needs to be looked at closely. For instance, medical teams in Bhopal have used a variety of procedures, tests and equipment which were primarily designed for clinic conditions. How reliable is the data they generate in field conditions? More fundamentally, what is the relevance of these data in the absence of an epidemiological profile? The fact that epidemiological methods were not used is itself a comment on the objectives and design of the study.

Another illustration is again from the U.S. A parish priest going on information gathered from mothers in the area came to the conclusion that an increase in the number of children with leukaemia was attributable to chemical dumping in the area. The state authorities made note of complaints but instituted no large-scale studies. Instead every time the community made complaints about a fresh incident of chemical dumping, the state authorities would arrive on the scene with new types of equipment, many of them being 'tested' for the first time, and would invariably discover 'no unexpected readings'. The preoccupation with quantification and the use of equipment served to divert attention from the real situation, which was the rising incidence of leukaemia.

One must also recognise the limits of scientific methodology in social studies. For instance, the concept of 'statistical significance' depends on the level of existing knowledge/information. And this acquires importance when study populations are being compared with loosely defined 'control' groups. In Bhopal since the levels of exposure were not available, the definition of what would be the control group itself came into question. This lack of background information became especially important when trying to determine say, pregnancy outcome or even for that matter gynaecological effects of exposure. The design, methods and material of the study reveal much that the stated objectives might seek to obscure.

## iii. 'Gut feeling' Vs. Scientific Objectivity

It is becoming increasingly obvious that because certain kinds of information are not amenable to quantification or measurement, they are conveniently ignored by studies and left out of the scope of investigations. Given the fact that there is a dearth of information linking pollutants to health status, it is often the 'gut feeling' of the victims which gives the first picture of cause and effect. But the subjective perceptions of the victims become important only in studies conducted by people who are affected. To an extent then the lack of appreciation of these unquantifiable, unmeasurable perceptions of the victims is indicative of a certain bias and must be noted when the conclusions of studies are being utilised.

## iv. Control of information and distortion of facts

This is perhaps the most obvious of biases. Every one of the studies mentioned earlier have resorted to choosing certain data in order to distort the conclusions. Published report of studies often do not reveal all the data which has been obtained in the course of the study. Thus access to primary information is necessary even to determine the validity of the conclusions of any study.

## Features of a People's Study

What might be the basis of people's studies as against the "experts' studies"? (1) Collective generation of hypothesis of the study. This ensures that the collective perception of reality gets emphasised and reduces 'experts' bias (2) Subjective feelings, symptoms and personal histories should be the primary data. Within the perspective generated from this data further clinical, medical and technical investigations may be carried out. (3) A thorough examination of supposedly 'objective' scientific methodologies and materials to be used, from the point of view of validity and relevance to the study on hand, (4) Integration of experts' opinions with the people's perspective after the evaluation. In other words, not taking the experts for granted and (5) An acceptance and realisations that the solution to these problems may lie quite outside the

narrow sphere of the workplace or the community concern and a commitment not to be satisfied with limited solutions forever. (This is based on the approach derived by the Italian Workers' Committees established after 1969).

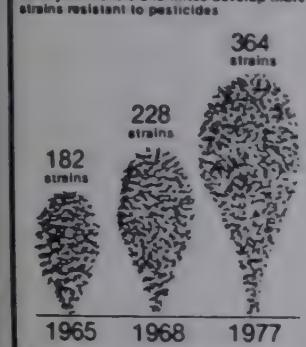
#### Bibliography

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- 5 'Workers participation and control in Italy: The case of occupational medicine' Giorgio Assennato and V Navarro in *International Journal of Health Services*, 10 (2) 1980.



### Resistance to pesticides

#### GROWING RESISTANCE



#### THE SUPER-PESTS



#### ATTACKING THE SUPER-PESTS

If a pest develops resistance to a pesticide there is a range of options

##### CHANGE THE CHEMICAL

by using other pesticides



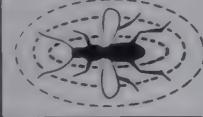
##### CHANGE THE ENVIRONMENT

By digging up egg pods for example



##### GENETIC CONTROL

by releasing male pests sterilized with X-rays to compete for females



##### BIOLOGICAL CONTROL

By releasing a natural enemy of the pest to destroy it



##### BEHAVIOURAL CONTROL

By attracting or repelling pests with sex hormones or chemicals



##### RESISTANCE BREEDING

by developing plant varieties resistant to pests



Each of these options presents problems. Great care must be taken in the choice of method adopted if pests are not to become even more resistant.

UNEP/Peter Sullivan

#### You could be a victim

if you work in or live near a factory producing or using hazardous substances.\*

- More than 100 plants in India use, store or make poisons as toxic as phosgene and cyanide.
- Over 500 factories handle chemicals proved to be cancer-causing.

You could also be a victim of invisible poisoning due to pollution. Many industries spew out slow killers such as oxides of sulphur and nitrogen. Their concentrations in our industrial areas are among the highest anywhere.

\* highly hazardous industries include dyes, plastics, paints, petrochemicals, pharmaceuticals, asbestos products, pesticides, industrial gases, acids and solvents.

poster from 'no more bhopals' - a ced exhibition

The aim of this paper is to highlight some of the important factors which modify the results, interpretations and conclusions of some of the available studies on pesticide poisoning. It is also an attempt to give reasons why such factors operate and to note the possible broader socio-political repercussions of such studies.

The scope of this paper is limited to giving the required pre-requisites for such studies and to show where some of the available studies vary from these criteria. It has not been possible to make an extensive comparative study of studies conducted by various agencies in similar situations. Hence any conclusion from this evaluation should be made with due caution.

#### General Recommendation for Pesticide Research

##### A. Occupational Health of Workers

The study should:

1. Place greater emphasis on the occupational health of workers.
2. Define the scope of health problems. This should be done through surveys determining
  - a. types and amounts of pesticides used
  - b. current state of health of workers in relation to pesticide exposure
  - c. the number, sex and age of workers exposed

##### B. Criteria for valid Epidemiological Studies

Epidemiological studies should include atleast

- a. environmental monitoring
- b. biological monitoring
- c. evaluation of health effects

Health-effects data obtained in a clinical setting must be well documented and supported with detailed occupational work histories.

##### C. Metabolic Studies

Studies of percutaneous fractional absorption should be made, taking into account vehicle, sites of exposure, sex and age. It is necessary to identify metabolites that are suitable for use in screening of exposed workers and analysed quickly under field conditions and are quantitatively related to airborne concentration of pesticides.

The effects which must be considered as adverse to the body must be well defined as follows:

- a. Effects that indicate early stage of clinical disease
- b. Effects that are not readily reversible and indicate a decrease in the body's ability to maintain homeostasis
- c. The effects that enhance the susceptibility of the individual to deleterious effects of other environmental influences
- d. Effects that cause relevant measurements to be outside the normal range if they are considered as an early indication of decreased functional capacity
- e. The effects that indicate important metabolic and biochemical changes

The WHO Technical Study Group recommends that the following criteria be used in the design and implementation of epidemiological studies.

##### A. Environmental Assessment of Exposure

###### 1. Method of sampling and analysis.

The study should contain information on

- a. type of sampling (personal or area)
- b. frequency distribution and duration sampling
- c. method of selecting group of workers for sampling
- d. detail on efficiency of sampling technique
- e. precision and accuracy of analytical methods used
- f. background concentration of pesticides

###### 2. Chronology

This aspect of the study should specify

- a. whether data reports to the time of study : are retrospective
- b. the relation between the exposure data and technological operations
- c. whether in course of time, changes in technology and for induction of hygienic measures could have changed the level of exposure

#### AN EVALUATION OF TWO OF THE AVAILABLE STUDIES ON PESTICIDES POISONING

Arvind M. Jha, Shriniwas J. Kashalikar and  
Kamalakar P. Kulkarni

### 3. Purity of agency and combined exposure

#### 4. Exposure history

The data should provide information

- a. past exposures or experiences with similar effects and .
- b. extent of total exposure through all routes

## B. Biological Assessment of Exposure

Special emphasis should be given to

1. the time of collection of biological specimen relative to period of exposure
2. reference levels in central groups as well as pre-exposure levels, if applicable.

## C. Evaluation of Effects and Response

### 1. The study should provide information on

- a. The exposure group - size, age, sex, socio-economic status, ethnic difference, medical history, duration of exposure, lifestyle of the person, in women-number of pregnancies, the number of abortions and other obstetric details
- b. The control group - same data as above
- c. Methodological aspects -
  - i. data-on-design
  - ii. the time of examination, objective interpretation of the effect, use of questionnaire in assessment of subjective symptoms and statistical methodology.

We are considering the following two papers for evaluation by applying the various criteria mentioned with foregoing account.

### Epidemic Malathion poisoning in Pakistan malaria workers

Baker L. Warren M. Jack M. Dobbin  
Dobbin R.D., Miles I.W. & Miller S.  
Alderman (1), Titters W.R. Lancet  
1, 31-33-1978

### Health hazard in pesticide formulators exposed to a combination of pesticides

S.K. Gupta, J.P. Jani, H.M. Sayed &  
S.K. Kashyap  
Ind. J. Med. Res. 79, 666-72-1984

1. Enough emphasis was given on the occupational health of workers by examining them and eliciting history for symptoms before and at the end of work on a typical working day. Technicians collected blood for cholinesterase activity. They also compared the health of the workers with that of those who used other pesticide namely DDT during this period.

Enough stress was given on the occupational health of workers and comparison was made with the health of control group engaged in similar physical work not involving pesticide. The clinical examinations and appropriate lab investigations were carried out, e.g. the whole blood, plasma & RBC cholinesterase activity was measured. Serum concentration of DDT, BMC & SGPT, SAP, Stalic dehydrogenase and s. proteins & A/G ratio were estimated. However, the paper doesn't clarify whether these estimations were carried out before and after the exposure on a giving working day.

2. The type and amount of pesticides was known. The exact dermal and respiratory absorption was estimated.

Exposure to pesticides were variable depending upon dust and liquid formulation which was used. There was no specific work as schedule exposure of workers was recorded in terms of weeks.

3. Due consideration was given to the current state of health of workers in relation to pesticide exposure

Due consideration was given to the current state of health of workers by assessing psychological, cardiological respiratory and gastrointestinal systems or by performing other investigations but dose-effect relationship was not established.

4. The sample included 425 spray men, 86 mixers & 95 supervisors. However, the age group was not mentioned.

The sample consisted of 96 male formulators but exact work schedule and nature of work is not described.

5. Air samples were obtained by standard methods to assess the respiratory exposure from airborne malathion. Skin patch testing for dermal exposure was performed and is very important.

The environmental monitoring was not done.

6. Biological monitoring has already been mentioned in point 1.

7. Details of health effects are documented

Details of health effects are documented but detailed occupational history is not mentioned.

Metabolites were not estimated.

9. The effects which don't lead to clinical disease are not considered.

The effects which don't lead to clinical disease are considered by studying SGPT serum, AP, LDH, proteins and A/G ratio.

10. Personal type of sampling not area.

Personal type of sampling and not area.

11. The method of selecting group of workers for sampling was well defined and accurate

Selection of workers was from unorganised sector with no specific work schedule hence ill defined and less accurate.

12. The authors considered two different methods for ACTM activity assay and selected the more accurate amongst the two namely MICELS Method.

Method of Voss & Sachsse using propionyl thiocoline as the substrate was used.

13. Background concentration was not estimated.

Background concentration was not estimated.

14. Use of protective devices if any is not mentioned.

It is clearly mentioned that protective devices were not used.

15. The authors point out to a reduction in incidences of toxicity due to good work practices and use of two pesticide formulation.

The authors suggested that health monitoring of workers may lead to reduction in incidents of toxicity.

16. Bulk samples of malathion powder used by spray team were collected & returned for analysis. The Inviko toxicity of pesticide sample correlated best with malathion content used by the team on the day blood samples were obtained. Samples of pesticides were administered to rats orally to determine LD50 values

Since the exposure was intermittent depending upon the availability upon the availability of technical material such work was not done.

17. Exposure history was well documented and extent of total exposure through all the routes was determined.

Exposure history was taken and classified as unexposed (controls) mild exposure (upto 1 mth) moderate (1mth-1yr) and heavy exposure (more than 1 yr) but extent of total exposure through all the routes was not determined.

18. Biological assessment of exposure was properly done by collecting biological specimen every day before and after the work. Reference levels in control groups were established.

Collection of biological specimen was related to exposure in terms of weeks. Reference levels in control groups was estimated.

19. The study gives information about size, age, sex, socio-economic status, ethnic differences, medical history duration of exposure, life style of the person.

The authors have given detail information on similar lines.

20. Appropriate statistical methods were applied for analysing the data and questionnaire was used (Student's t test & correlation co-efficient were used)

Appropriate statistical methods were applied for analysing the data and questionnaire was used.

## Motivational Aspects

For studies on pesticide poisoning the research team should have commitment towards people. Secondly, the team should have adequate training and technical knowhow. Thirdly, the team should have access to adequate source material. Fourthly, the members of the team should not have insecurity about their own status. Fifthly, members of the team should not have vested interest which are likely to distort the results and interpretations and lead to subsequent social and political repercussions.

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As mentioned in point

## The Politics of Chemicalisation

Every historical period is characterised by a dominant contradiction around which social transformation takes place and political conflicts get defined. The first industrial revolution was characterised by the social contradictions generated by mechanisation. In England and Europe, these contradictions were translated into the contradiction between labour and capital which Marx captured vividly and richly.

In colonies like India, which supplied raw materials for the Mechanised textile industry of Britain and whose indigenous textile industry was destroyed to create markets for mill produced cloth, the contradiction was translated into two conflicts. The first was between peasants wanting to grow food, and colonial powers wanting to grow indigo and cotton. The indigo revolt, the Champaran satyagraha and the Poona-Ahmednagar riots were political struggles emerging from this contradiction. The second contradiction was between the handloom producers of India and the mill owners in England. Gandhi's "Charkha" and the concept of "Khadi", was the political weapon of resistance against the domination of British mills over Indian handlooms.

Since the second World War, the primary source of global contradiction in economic processes has shifted from the mechanisation process to the chemicalisation process. The chemicalisation of health care, of agriculture and of manufacture creates a new political context in which the primary, though little recognised, conflict is between methods which are life-destroying, hazardous and violent on the one hand and life-enhancing, safe and non-violent methods on the other.

The myth of "a pill for every ill" which has been associated with the production and sales of hazardous and irrational drugs is being challenged all over the world. The 'pill' has proved to be a cause and not a cure, for disease and ill health.

Unfortunately, in agriculture, the myth that increased per capita consumption of pesticides is an indicator of progress goes unchallenged. Scarce capital is diverted to provide subsidies for increasing the use of chemical pesticides. The government's agricultural establishment thus acts as a sales representative for the pesticides lobby. This has destroyed ecologically stable and sustainable farming systems, and replaced them with cropping patterns vulnerable to pests, thus creating a self perpetuating market for hazardous chemicals. The problems relating to the chemicalisation of agriculture have been brought into sharp focus by Bhopal. It demonstrates the conflict between the people's right to life and survival and 'profit', the motive force behind increasing chemicalisation in all sectors of human life. It is this conflict, which is central to pesticide politics. Bhopal has forced a reassessment of the real costs and real benefits of chemical pesticides.

This paper attempts to analyze the violence and vested interest inherent in chemical pest control. In the next chapter, an analysis of how pest problems are introduced into agriculture and how pesticides fail to effectively control pests is attended. Finally, an attempt is made to show that safe, scientific and natural alternatives do exist and that hazardous chemicals are dispensable.

## Pesticides : Prescription for Fostering Pests

Traditional (or what the reductionist world view has labelled unscientific) systems of food production have managed pest control by a series of measures which include building up plant resistance, practising rotation, and mixed cropping, and providing habitats for pest predators in farms, trees and hedgerows. These practices

created a stable local ecology and economy. A balance was achieved between plants and their pests, through natural competition, selection and predator-prey relationships.

Folklore is generally found to be an important source of traditional knowledge about the ecological processes. For example, the Kayape Indians of the Amazon basin have a ritual in which the women paint their faces with ant parts in the maize festival. The principal theme of the myth is the celebration of the little red ant as a guardian of the fields and a friend of the women. This appears to be a baseless ritual from the reductionist point of view. But, as pointed out by Posey,

"the myth begins to make sense when we understand the co-evolutionary complex of maize, beans, manioc and this ant. Manioc produces an extra floral nectar that attracts the ants to the young manioc plant. The ants use their mandibles to make their way to the nectar cutting away any bean vines that would prevent the new, fragile manioc stems from growing. The twining bean vines are, therefore kept from climbing on the manioc and are left with the maize plants as their natural trellis. The maize can shoot up undamaged by the bean vines, while the bean plant itself furnishes valuable nitrogen needed by the maize. The ants are the natural manipulators of nature and facilitate the horticultural activities of the women".

'Scientific' farming upset this balance and created favourable conditions for the multiplication of disease. Organic fertilisers which build up plant resistance to disease were replaced by chemical fertilisers which decreased plant resistance to pests. Since many pests are specific to particular plants, the replacing of crop rotation by crop repetition encourages build up of pests. The substitution of multi-cropping methods by monocultures also makes the crop more prone to pest attacks. The mechanisation of farming also leads to the destruction of hedgerows and farm trees and thus destroys the habitat for some pest-predators.

The problem of pest control was therefore a problem created by the upsetting of the ecological balance of agro-ecosystems by the introduction of reductionist 'scientific agriculture'.

For reductionist scientists the problem is defined more in terms of the evidence of pests and not its ecology. Thus its only solution, which is favourable to the pesticide industry, is to create and sell poisons to kill these pests. As a pesticide company announced in a T.V. advertisement "the only good bug is a dead bug".

Commenting on this approach, De Bach says :

"The philosophy of pest control by chemicals has been to achieve the highest-kill possible, and per cent mortality has been the main yardstick in the early screening of new chemicals in the lab. Such an objective, the highest kill possible, combined with ignorance of or disregard for, on-target insects and mites is guaranteed to be the quickest road to upsets, resurgences and the development of resistance to pesticides".

De Bach's research on DDT-induced pest increases showed that these increases could be anywhere from 36 fold to over 1,200 fold.

The problem is thus magnified instead of being solved. The aggravation of the problem is related to the violence unleashed on natural enemies of pests. Reductionism has thus failed to recognise that these natural predators have the unique property of regulating pest populations and thus attaining a natural balance. It also fails to anticipate and predict what will happen when this balance is upset. Besides reflecting cognitive weakness, the approach of overkill generates violence in a number of other ways. More and more pesticides are needed to cope with the increasing pests. These chemicals are absorbed by plants and animals and enter the food chain and the eco system. Pesticides are thus becoming a

major source of water pollution and health hazards. Rachel Carson's "Silent Spring" remains the best commentary on this aspect of violence.

The introduction of pesticides is largely a function of exaggerated claims of the "damage prevented by scientists and industry". Natural enemies work quietly, invisibly. On the other hand, "the effects of chemical pesticides are highly visible and quickly publicised. Dead bugs are an impressive sight and a good 'selling point'." The drama of violence thus distorts and misguides the criteria for effectiveness. This illegitimate translation of violence as a sign of effectiveness establishes the myth of the cognitive superiority and success of a modern reductionist science.

The mystification of violence as control runs throughout the scientific process, beginning with the concept of the 'scientific' and 'controlled' experiment.

Experiments comparing the natural control of pests to the chemical control of pest are not real life experiments. The very concept of natural or biological control is ecological and it entails sub systems which are impossible to create in so-called experiments. Usually the test plot not treated with pesticides is surrounded by plots treated with different materials, dosages, and intervals of application. The untreated plot is therefore affected by chemicals from its surroundings as well as the effect of the earlier sprayings. The natural enemies on this sample plot are thus destroyed. Pests tend to explode on this check plot. Meanwhile the check plot treated with pesticides shows a greater amount of "damage prevented". The "controlled experiment" in pesticide research has the in-built tendency to under-estimate the potential of natural control and inflate the promise of chemical control of pests. These exaggerated figures for crop losses have led to tall claims such as those of DG Hessayan, of the British Agrochemicals Association who said that "the effect of not spraying tropical crops would be disastrous, and the resulting famine would be the greatest disaster the world has ever known.

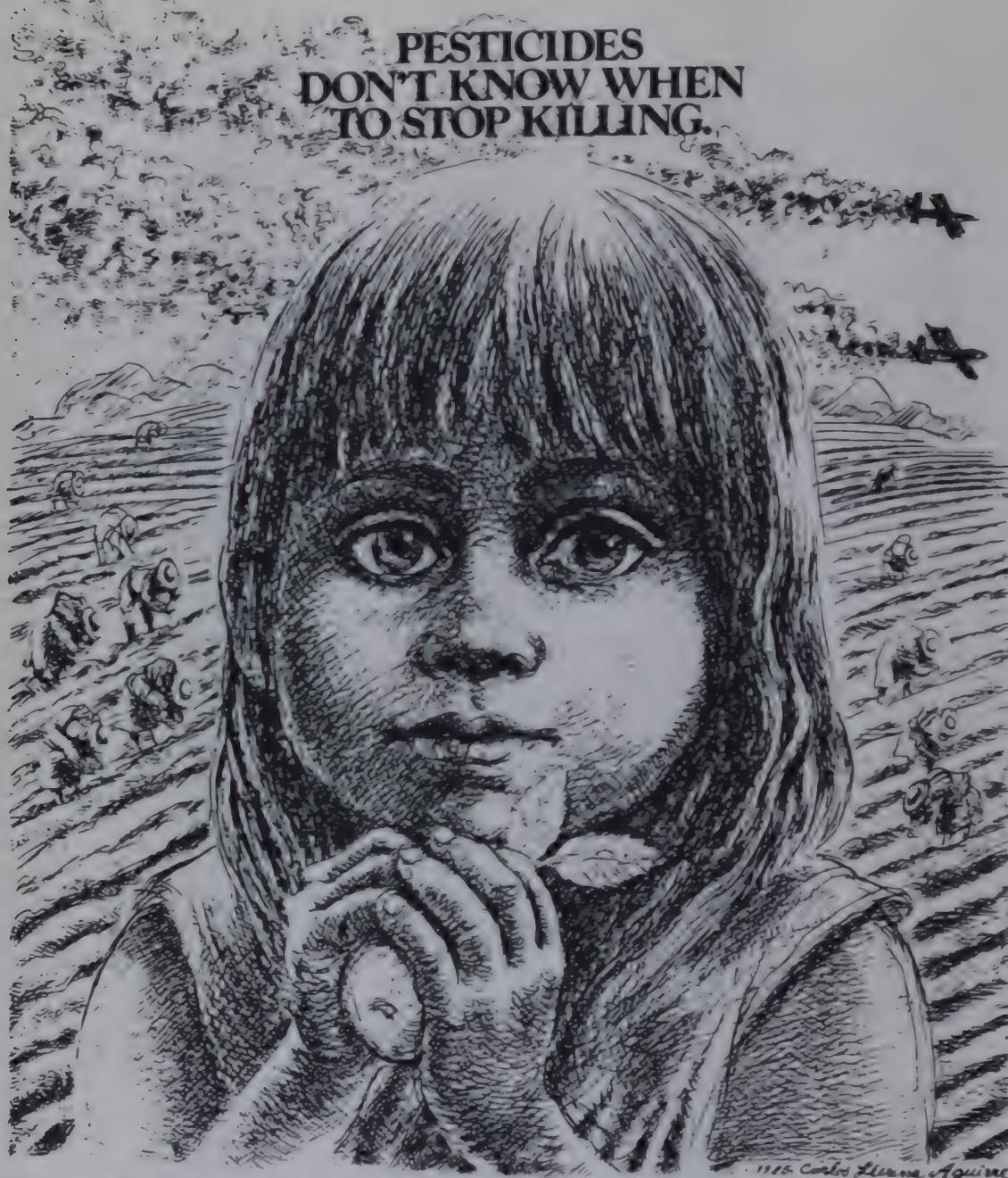
The scientific test also tends to wrongly assess ill-effects of pesticides on the environment and on human health.

Testing of pesticides is carried out primarily on the basis of acute toxicity, (that is, effects from short term exposure and comparatively high doses) ... It is fine to test for acute toxicity, but what if there are undetected effects on humans, effects that may not show up for many years or generations? Our limited testing is unlikely to indicate this. Cancer may take 20-30 years to appear. Besides, animal experiments are especially unreliable in tests for cancer, mutations and allergies which are less transferable between species than acute toxicity ... The testing procedure also neglects a number of other factors including the possible interaction between residues and other food additives, hazards of metabolic products, increased susceptibility of the young, and the possible adverse nutritional effects of induced metabolic changes in food. These possibilities indicate increased risk to consumers and to society from the continued use of chemical pesticides. (Ignorance, irrationality and greed for profits are the characteristics of the pesticide sector, as has been most dramatically revealed in the Bhopal disaster. Union Carbide was simultaneously a creator of scientific knowledge, profits and violence)

Inspite of complete failure to solve the problem of pest control and inspite of the destruction and violence against nature and man, pesticide sales continue to go up. Their use is insured by the state's agricultural policy and pesticide subsidies and through pesticide propaganda. Pesticides are now "ecological-narcotics" - and their usage has become habit forming, and seemingly essential. The continued use of pesticides would destroy species of pest-predators, thus consigning the potentially more effective biological control methods to the museums by destroying the ecological basis of alternative systems of pest management.

And this alternative system is being undermined and destroyed because it does not have a potential for profits. Natural enemies and natural pesticides are not marketable! Moreover, biological control methods or natural pesticides would not be profitable because they

are ecosystem specific, whereas pesticides are chosen for their "broad-spectrum" capabilities to work across and against ecosystems. They have a large market potential. Science, violence and profits seem to be inextricably linked to one another in the reductionist model of modern pest control. Violence is not an indicator of control. On the contrary it is a sign that the system is beginning to get out of control.



## FIGHT THE DIRTY DOZEN WORLDWIDE

HCH/Lindane · Camphechlor (Toxaphene) · Chlordane/Heptachlor · Chlordimeform (Galecron) · DBCP · DDT  
Aldrin/Dieldrin/Endrin (The "Drins") · EDB · Pentachlorophenol (PCP) · Paraquat · Ethyl Parathion · 2,4,5-T

**PAN** Pesticide Action Network (PAN) International

Produced by Public Media Center.

A false dilemma that is frequently posed is that we must either live with the hazards of chemical pesticides or with malnutrition and starvation.

To us, the real choice is between sustainable farming systems that maintain high productivity and pesticides which create pest problem and threaten a higher incidence of crop failure. Pesticides are a sure way to famine since they work against the basic biological productivity and living principles of crop production.

The productivity breakdown happens in the following chain -

indigenous pest resistant varieties  
are replaced by  
hybrid strains which are vulnerable to pests and disease  
creating the need for  
increased use of pesticides  
which causes  
pesticide resistance, pest outbreaks  
which leads to  
crop failure

Ironically, this collapse in productivity is a result of a chase for increased productivity. To illustrate how this cycle works, we will take the case of two of India's oldest field crops which have had stable production for centuries but are now facing the threat of increased crop failures:

#### The Pest Problem in Cotton

Cotton varieties in India belong to four distinct species namely Gossypium arboreum, G.herbaceum, G.hirsutum, G.barbadense. The first two belong to the old world and are generally called 'desi' cottons. The hirsutum and the barbadense cottons are generally known as American cottons.

Cotton probably has a long and complex history. India had a flourishing export trade in cotton and cotton piece goods from as early as 500 BC till the early part of the nineteenth century. With the Industrial Revolution in Europe and the consolidation of British power in India, emphasis shifted from the manufacture of fabrics to the production of raw cotton for supplying the Lancashire mills in England. In fact, the introduction of American cottons in India can be traced back to the early history of the British East India Company. Since then efforts to "improve" cotton have been continuous. Some of the earliest breeding work was done in Punjab. Dharwar district in Karnataka was also an important breeding centre for Indo-American varieties.

After the partition in 1947, cotton was given top priority by planners and agriculturists because nearly two fifths of the cotton cropping area was lost to Pakistan whereas 95 per cent of the mills remained in India. The government launched a massive 'grow-more-cotton' campaign in all the major cotton growing states from 1950-51. The idea was to increase the production as rapidly as possible by expanding the area under cotton. All legislative curbs imposed on cotton cultivation during the second world war when the accent was on food were suspended and incentives like irrigation subsidies on inputs and loans and good seed loans were provided. Remission of land revenue was also granted to cotton growers. The government even guaranteed to make good any shortfall in foodgrains caused by diversion of land from food-crops to cotton.

This horizontal development resulted in a spurt in production from 30.4 lakh bales in 1950-51 to 41.81 lakh bales in 1955-56. The sixties saw vertical development as the effort was changed to produce more bolls

#### **PESTICIDES V/S FAMINE THE FALSE DILEMMA**

Mira Shiva and Vandana Shiva

per plant. The average production in the sixties was 52.66 lakh bales against 40.8 lakh bales and the productivity sharply rose from 95 kg/ha to 113 kg/ha. However the demands of cotton mills in the country were constantly increasing. The government then launched 'package programmes' for intensive cultivation in 1962-63. It offered to bear 50% of the cost of production. Yet the production fell short of target due to crop failures. In 1971 the Intensive Cotton District Programme (ICDP) was implemented in 14 key cotton growing districts. The thrust of the programme was on irrigated cottons and relied heavily on high yielding varieties. The main objective of the programme was to sizeably increase production and wipe out the existing deficit as quickly as possible. The production shot up to 69.50 lakh bales during the first year but slumped to 57.35 lakh bales in 1972-73.

The government had offered all sorts of concessions and benefits to the farmers. Punjab was one of the first states to benefit from the programme. The following list of assistance items gives a fair idea of the kind of investment made by the government:

ITEMS	ASSISTANCE
Aerial spraying	25% of charges
Insecticides for six sprays	Rs. 50 per hectare
Plant protection equipment	25% of costs
Weedicides	33.3% of costs
Cotton seeds	25% of cost
Inputs for demonstrations	Rs. 400 per hectare

The government still believes that the ICDP will boost the cotton production in India to a higher and more sustainable level and is busy extending it to newer areas. By increasing the area under hybrid cottons from 7.5 lakh hectare to 11.5 lakh hectare in the next five years, the government intends to step up the production of cotton in the country. This would also mean a corresponding increase in irrigation, fertiliser application, plant protection and improved seeds. According to the Indian Ministry of Agriculture the total cotton production should be about 96 lakh bales by the end of the VII Five Year Plan (1984-85 to 1989-90) i.e. a 4% increase in production in the next five years.

However, reports from all over the country show a completely different trend. The area under irrigate cotton is actually on the decline and peasants are showing preference for more reliable food crops. In Punjab and Haryana where a large proportion of crop land has been brought under irrigation, the share of cotton in the gross irrigated crop-area has reduced. A recent report from the Gujarat Agricultural University says that the total area of cotton in Gujarat has reduced from 17.10 lakh hectares in 1959-60 to 15.72 lakh hectares in 1980-81. Other areas in India have also shown a similar trend. In Tamil Nadu, one of the major cotton growing states, some 43,000 ha were under irrigated cotton in 1957 but the area is rapidly shrinking as farmers have begun to realise that growing paddy is more profitable. In Tamil Nadu, cotton is grown all round the year and this uninterrupted pattern is extremely favourable for pest build up. As a result, pest control costs have become prohibitive for most farmers. The same thing seems to have taken place in Karnataka. The area under hybrid cottons reduced from 97 ha to 85 ha and was taken over by groundnut and jowar. The farmers of Krishna district in Karnataka also prefer growing high yielding rice varieties rather than the popular P216 cotton which requires far more care and is expensive to grow. In the Yeotmal and Akola regions of Maharashtra farmers are switching back to Jowar which is cheaper to produce. Others are going back to older varieties of cotton which need less fertiliser plant care. For example in 1975-76 several farmers in Raichur district of Karnataka went back to 'Jaxmi' and 'Jayadhar' instead of the newer 'Varab' about Rs. 2,000-3,000/acre for production and the returns were only about Rs. 600-800 per acre. In the Khargone region of Madhya Pradesh too the farmers have switched over from H4 to older cultivars like Khandwa 2 and A5-9 though they have by and large maintained the acreage under cotton.

In some parts of Punjab the change has been more drastic. Some farmers have actually gone back to 'desi' varieties to minimise risk and investment.

### The Pest Problem

One of the major problems of cotton cultivation in India is the large number of pests which attack the crop. In 1948 as many as 109 pests were found to infest cotton crops in the country and the number seems to be increasing every year. Crop failures due to pest attack are not uncommon. According to Paushak Ltd, a Gujarat based pesticide manufacturing firm, the annual loss of cotton crop due to pest attack alone is to the tune of 50 million rupees.

The absolute damage caused by pests to cotton also seems to be increasing every year. In 1925 at a joint meeting of the Agricultural, Zoological, Botanical and Medical research stations of the Indian Science Congress held in Banaras, the losses caused by the pink bollworm, one of the major pests, were put at 25 per cent. The Gujarat Agricultural University progress report for the last eighty years says that damage by pests to cotton can be as high as 96.7 per cent of which bollworms are responsible for 74 per cent. Most of the damage to the crop is done by bollworms and jassids. Bollworms attack the cotton flowers and bolls at all stages of growth. Younger bolls are shed when attacked and older ones produce cotton of inferior quality when infested. The most important pests of cotton in India are:

Pink bollworm	Adult and Larva	<u>Pestinophora gossypiella</u>
Spotted bollworm	Adult and Larva	<u>Earlas spp</u>
American boll-worm	Adult and Larva	<u>Heliothis armigera</u>
Jassid		<u>Emrasca devastans</u>
Aphid		<u>Emrasca bygutulla</u>
		<u>Aphis gossypii</u>

'Scientific experts' have created unreal conditions for stepping up the productivity of cotton. Areas which were earlier inundated only by annual rains are now constantly under water from irrigation canals. High yielding varieties producing long stapled cotton have replaced the sturdy desi varieties. In some places, cotton is grown all the year round whereas traditionally the peasants took only one or two crops of paddy in a year. All this has disrupted the ecological balance. Human beings have reversed the pattern of nature or they have modified the environment for the crop. The spreading incidence of pests on cotton has been closely linked with the spread of irrigation, high yielding varieties and intensive cultivation practices.

Prior to 1903 bollworms were not even considered a serious problem in India. In 1905 the cotton crop in Punjab and Sind failed and scientists attributed this to bollworm infestation. In South India too, bollworms became a menace after the introduction of American cottons and the whole package of cultivation practices which came with them.

Jassids have a similar history. They came into prominence in the Punjab about 85 years ago when the American cottons began gaining ground. American cottons in the Tungabhadra region of South India are also prone to jassid attack.

The strategy adopted by agricultural scientists to fight this growing problem was to use "plant protection" chemicals. It is estimated that something like 60% of all the pesticides used in the country are for protecting cotton alone.

But pesticides also kill natural predators and parasites of the pests and this factor has also led to an increase in pest populations. Insects which though present earlier were never classified as 'pests' have now assumed that status as their population have increased drastically in the absence of any natural enemies. For example the cotton leaf roller which was never a serious threat to cotton suddenly caused noticeable damage to the crop in U.P. in 1895, 1900, 1927, 1942 and 1945. In 1964 cotton stem weevils Aleiodes affaber appeared as a significant problem in Gujarat on Indo-American cottons. In 1977 the cotton bud moth which was earlier a minor pest became a serious pest in the western cotton growing tract in Punjab. Similarly in 1978 Heliothis assulta, another

variety of bollworm, was recorded for the first time as a pest in Haryana. In Muktsar district of Punjab Heliothis armigera became a serious pest on the cotton crop because of a shift in the cropping pattern, absence of any natural enemies and wet soils in 1980.

But this is not all. Pests also develop resistance to pesticides. So the pesticides have to be modified frequently to keep up with the changes in pest populations. At the farmer's end there is total confusion and chaos. Take for example the spraying schedule for H4. According to the world bank document, farmers in several parts of India were spraying too much and too soon. According to G.C. Verma and Maninder of the Punjab Agricultural University, 5-6 sprayings with DDT, Fenitrothion, Endosulfan, Carbaryl, Quinalphos, Monocrotophos, Phenthroate and Phosalone are common practice. The farmers in Ferozepur, Faridkot and Bhatinda (among the main cotton growing regions in Punjab) have been using insecticides indiscriminately for the last 10-15 years. Cotton cultivation in these areas seems to be on the verge of collapse. These two scientists fear that unless something is done quickly, the situation will become like the one in the Canate valley of Peru in 1969 where cotton cultivation became totally uneconomical due to increasing costs of plant protection. As Smith and Van den Bosch write:

The Canete story stands as a classic example of the problems that can beset pest control which ignores ecology and relies on unilateral use of broad-spectrum insecticides."

In Mexico too, pesticides use has destroyed cotton cultivation. Between 1960 and 1970 the area under cotton in the Matamoros-Reynosa belt declined from 7,10,000 acres to 1,200 acres. While this was happening, 5,00,000 acres were opened in 1966 for cultivation in the Tampico-Mante area. By 1970 only 1,200 acres were left. This is attributed to the increase in the intensity of pests, particularly the tobacco budworm, which is an upset species, and the inability to cope with them through unilateral use of chemical pesticides in spite of attempts to increase dosages and frequency of application. Widespread unemployment and economic ruin were the result. As Adkisson has said "The seeds for the destruction of the Mexican cotton industry were planted when the producers decided that their insect pest problems could be best solved by the unilateral use of regularly scheduled applications of broad spectrum pesticides."

### The Pest Problem in Ragi

Ragi (*Eleusine Corocava*) is one of India's staple course grains. Introduced from the Horn of Africa more than 40 centuries ago, it has provided a balanced diet to Indian peasants over millenia. It is a miracle crop - very hardy and drought resistant. Even under unfavourable conditions the crop maintains its growth. It is remarkably free from fingers and pest attacks. It is stored in underground pits and can keep for years. It has been found that the protein of ragi is biologically complete as in the case of milk. Writing in 1886, Church noted that ragi "is a fairly productive rainy-weather crop for light soils. It may be grown almost upon stones and gravel. It yields from 5 to 6 maunds of grain per acre upon the hills, 12 to 14 maunds in the plains, if carefully cultivated and weeded."

A better mix of desirable properties: highly nutritive, highly productive, pest-resistant, draught resistant could not be found. Yet the crises mind could not resist trying of 'improve' ragi.

New varieties were introduced in 1960. The All-India Co-ordinated Millet Improvement programme developed the IE variety and the Indo-African variety called INFAF. Table I gives the performance of indigenous and HYV's ragi during 1976-77 and 77-78:

	Ragi	HYV ragi		
	1976-77	1977-78	1976-77	1977-78
Area (ha)	132439	125259	53078	90531
Production(mt)	124176	149983	60133	129396
Yield (kg/ha)	938	1197	1138	1429
Average		1022		1283

The HYV's performance does not excell the indigenous varieties' performance significantly. Infact, under stable cropping, as in Church's data, indigenous varieties had yields as high as that of HYV ragi. But there are new costs and risks introduced.

The new INDAF varieties are not drought resistant like the older varieties. The main problem with these varieties is the damage done to growth in the absence of rainfall during critical stages. If rainfall is scanty, the expected yield is neverneared. The local varieties does not fail as much as the new varieties in the event of late rainfall during the tillering stage. Also the INDAF varieties are amenable to certain pests and diseases and if no timely plant protection measures are undertaken, the crop would be a failure. Plant protection chemicals are sprayed 2-5 times for irrigated and 1-2 times for dry ragi. In Dharmapuri in Tamil Nadu, because of uncertainty connected with the HYV's, locals are being preferred due to their "guaranteed yield" and lower costs. The additional fertiliser and pesticide cost per ha. of HYV ragi are Rs. 250 approximately. This is a guaranteed cost. This is off set only in the best years. In years of rainfall fluctuation or pest damage, the yield and incomes from HYV ragi are much lower than they are from local varieties. The 'improvement' in ragi has merely managed to change a draught and pest resistant crop into one that is vulnerable to rainfall variation and pests. With the HYV package, including pesticides, are sown the seeds of famine. The choice is not between pesticides or famine. It is between ecologically stable and sustainable food production which does not need pesticides and ecologically unstable and unsustainable cropping systems which used pesticides but cannot prevent crop failure.

Perfect ecological balance would not allow pests, because there would be a pest-predator balance. This, of course, is the ideal in food production. But even if we do not achieve the ideal, and pests do occur, poisons are not the only alternative in pest control. Nature has endowed us with safe natural pesticides which are far superior to manufactured poisons. It is to one such safe yet powerful 'natural' pesticide that we will now turn.

### The Safe Alternative

#### Neem : The Natural Pesticide

When our mothers and grandmothers wanted to preserve foodgrain or clothes, they did not use toxic, hazardous chemicals. They used the dried leaves of the beautiful neem tree (*Azadirachta indica*).

Different parts of the neem tree have excellent 'antifeedant' repellent and insecticidal properties. The most important development in recent years has been the finding regarding hormonal effects of some neem products causing disturbances of metamorphosis of insects.

Seeing the promise of the pesticide properties of Neem, a global scientific research programme has been launched. In Germany four research teams are actively working on Neem. In America, the USDA has been participating in the neem research programme since 1980. It is collaborating with State Agricultural Experiment Stations, universities, growers and industry, and has reached the point at which a commercial concern in the United States is actively pursuing the development of neem as a practical marketable pest control agent.

Cherry trees affected by the Japanese beetle in Washington D.C. were sprayed with a 1% crude ethanol extract of neem kernels. The damage was held at 2.4% (leaf loss on side branches) as compared with 35.5% loss on nearby nonsprayed trees.

When a suspension of neem kernel as low as 0.05% was sprayed on different crops which were then offered to the desert locust, the pest preferred to starve to death rather than feed on treated leaves. In 1962, when the farm of IARI was attacked by the locust, no damage was done to the crop since they had been sprayed with 0.1% neem kernel suspension. Tobacco crops have been found to be effectively protected from the tobacco caterpillar with a 2% neem kernel suspension in water.

The efficacy of neem products, particularly 3 and 5% extracts of neem kernel and neem oil, was determined against *Heliothis armigera* in multilocation trials with chickpea carried out under the All India Co-ordinated Pulses Improvement Project.

The result of trials on antifeeding and other properties of neem kernel have proved beyond doubt the utility of neem in controlling insect damage according to IARI.

IARI has also scientifically tested the efficacy of powdered neem kernel in protecting stored grain (wheat, green gram, chick pea, peas etc.) against pest damage. No egg laying was recorded on treated seed and therefore population build up is nil. It has been suggested that oil present in the powdered neem kernel may act as a protective coating, because of which the insects do not deposit eggs on the grain. Antifeedant effect and inhibition of over-position are other possible mechanisms by which the grain is protected by neem seed components from damage by different insect species. Neem also controls pests through hormonal effects causing disturbances of metamorphosis of insects. The azadirachtin disrupts the co-ordination of endocrine functions.

Juvenile hormonal effect has been observed in *S. litura*, *C. partellus*, and *H. armigera*. Either the larvae died during the process of development, or the adults emerging from the surviving larva were abnormal and did not live long enough to produce another generation.

Neem is a genuine people's pesticide, safe, indigenous, and effective. It does not require particular conditions of climate and soil, and grows from Rajasthan to Bengal, from Punjab to Tamil Nadu. According to a KVIC estimate there were 1,38,99,067 neem trees in India in 1976, with a potential of yielding 4,18,633 tons of seed per year. If afforestation schemes of social forestry and wasteland development are used to propagate neem instead of Eucalyptus, the Indian peasant and tribal could be the owners and producers of safe pesticide plants. This is the contemporary alternative to the Union Carbides and the inevitability of Bhopals.

Neem differs from chemical pesticides in two important ways. Firstly, since neem acts on a number of behavioral and psychological processes rather than physiological functions of the insects, the chances of build up of resistance in target insects is low. Secondly, while poisonous chemicals kill insects as well as humans and species useful to man, neem is not hazardous to warm blooded animals. It is remarkably harmless to vertebrates. Cattle have been fed neem oil cake, people have used neem sticks for dental care (datun) and neem leaves and flowers are also part of the human diet. From the health perspective, neem is a miraculous alternative to poisons. It not only controls pests, but also has medicinal properties.

The seed yields margosa oil, which is used in ayurvedic and unani medicine for skin diseases. When planted around villages, it is a prophylactic against malaria. Recent research has shown that neem leaf extracts have a promising future as a vector control agent in the public health to save human populations from diseases spread by mosquitoes.

#### Pesticide Action as Health Action

Public education on health hazards of pesticides will leave people passive as long as they believe to the false choice of poisons v/s famine. Counter-campaigns on sustainable agriculture, and natural pesticides are important if timely action has to be taken. The Bhopal disaster has opened our eyes. The best guarantee towards prevent-

ing future Bhopals is to promote safer alternatives in food production and safer ways of dealing with pests. These are issues in health care.

Two actions that immediately come to mind are

1. The conservation and propagation of native varieties of food crops which are nutritive, drought resistant and pest resistant such as ragi.

2. The conservation and propagation of natural pesticides such as neem which are safe and effective, and have multiple use in health care and basic needs.

3. Ongoing afforestation programmes such as 'social forestry' and 'wasteland development' should be used to build these alternative pesticide factories, which do not kill, but heal both people and plant.



"We ordered mussels, not DDT, PCB, petroleum, plutonium and lead!"

©Earthscan 1984

Cartoon by Nikki Meith

The poisonous effects of pesticides on human beings is undisputed. Out of the innumerable number of chemicals being used for the production of pesticides and insecticides, a large number have been tested for carcinogenicity. Most of the pesticides have been banned in countries of their origin - USA and W. Germany. Those few which are allowed are regulated by stringent laws. Despite their known harmful effects, India not only continues to manufacture pesticides but also imports large quantities from the very countries where they are banned. Instead of banning or restricting its use, India's demand and use of pesticides has been rising dramatically. Majority of the pesticides used in India are either banned or highly restricted in the Western countries.

What is frightening is that despite the massive expansion of the petro-chemical industry and the large-scale importation of the pesticides into the country very few laws exist to restrict and regulate the use of pesticides. And those which exist are rarely enforced. The only solitary law dealing with pesticides is the Insecticides Act, 1968. However, there are some general acts which would apply to the manufacture and use of pesticides. We shall classify these laws into three types.

- a. Laws relating to the working conditions at the work place.
- b. Laws dealing with environmental pollution including food contamination.
- c. Civil liabilities under the Law of Torts.

#### Laws relating to the Working Conditions of the Work Place

As mentioned above, there are no special laws dealing with pesticide manufacture and its harmful effects on workers. However, all employers are obligated to provide safe and healthy work places for their employees. The most important acts which lay down these obligations are the Factories Act, 1948 and the Insecticides Act, 1968. Two other acts which in particular deal with the payment of compensation for diseases contracted at the work place are the Workmen's Compensation Act, 1923 and the Employees' State Insurance Act, 1948.

1. The Insecticides Act, 1968 was enacted with the object of regulating the import, manufacture, sale, transport, distribution and use of insecticides with a view to prevent risk to human beings or animals. Though the act is comprehensive it has failed in achieving its objective. The task of the Central Insecticides Board constituted under the act, is to advise the central government in matters relating to, amongst others, the manufacture, sale, storage, transport and distribution of insecticides "with a view to ensure safety of human beings or animals". Though the functions of the Central Board are merely advisory, the Registration Committee appointed by the central government under Section 5 of the Act is entrusted with the task of registering insecticides in accordance with the provisions of the act. The Central Board is empowered with the authority of making bye-laws for the purpose of regulating its own procedure. Thus, if any person desires to manufacture or import any insecticide he/she must seek the sanction of the Registration Committee. The Committee may refuse to register the insecticide if it is of the opinion that the use of the insecticide involves serious risks to human beings or animals. The guidelines as to what constitutes "serious risks" are not laid down. However, the application must be considered within a period of 12 months which will be extended by a period not exceeding 6 months. Where the Committee is of the opinion that the insecticide is being introduced for the first time in India, it may provisionally register it for two years. Any person desiring to manufacture or import any insecticide is also required to obtain a licence from the licensing officer which is to be renewed from time to time. Thus the act lays down an elaborate procedure for the granting of registration certificate and licence. Section 16 of the Act sets up a Central Insecticides Laboratory. The central government or the state government also has the power to appoint insecticide analysts and insecticide inspectors possessing the requisite technical knowledge

## **REGULATORY LAWS RELATING TO THE USE OF PESTICIDES IN INDIA**

Gayatri Singh

and qualifications. All cases of poisoning after being notified in the official gazette proceedings against any company or person for contravention of any of the provisions of the act are to be held before a magistrate. If it is found that the company has violated the conditions attached to the licence, the safety regulations and the maintenance of inspection records, it can be prosecuted under Section 29(3) of the act. Its registration number can be confiscated. Under the act, it is no defence to say that the accused was ignorant of the nature or quality of the insecticide or the risk involved. Hence, it would seem that the manufacturers and the importers would be strictly liable for any offence under the act. In respect of workers handling and manufacturing insecticide the central government may prescribe such rules relating to the protective clothing and equipment to be used by workers and the manner of their use by the workers; the precautions to be taken against poisoning through the use or handling of insecticides; the instruction and training to be provided regarding the use of things supplied to the workers for ensuring their safety; and the facilities for medical examination of the workers engaged in the manufacture of the insecticides.

#### Critical Comments

Despite the elaborate and intricate procedure laid down for the detection of hazardous and poisonous substances, hardly any companies manufacturing insecticides have been prosecuted. The coterie of inspectors and analysts are inexperienced and corrupt. The facilities to test and monitor the nature and quality of insecticide is inadequate. There is no central monitoring system. The considerations and licences are not known to the public. The records are not open to public scrutiny. Similarly, the reports of the inspectors and the analysts are not easily accessible. The workers are not informed about the nature of the insecticides they are handling. In fact, the Garg Committee appointed by the Pollution Control Board, in its report, stated that most of the multinational companies were openly flouting safety regulations within the factory. Most of these companies were those manufacturing pesticides and insecticides. The central government has miserably failed to prescribe any rule worth its name for the protection of workers handling or manufacturing insecticides. Thousands of workers continue to die due to exposure to dangerous chemicals used for the manufacture of pesticides. One major drawback of the Insecticide Act is that prosecution for any offence under the act "can be instituted only by, or with the written consent of the state government or a person authorised in this behalf by the state government". All power is concentrated in the hands of the central and/or the state government. Moreover, the accused may be acquitted and exonerated of all charges if he can show:

- a. that the offence was committed without his knowledge, or
- b. he exercised all due diligence to prevent the commission of such offence.

#### Some tentative suggestions to make the Act more effective

- a. The criteria for deciding the toxicity of a substance and for granting a registration certification to importers should be based on whether the country importing the pesticide has banned it within its country or not. The country banning a particular pesticide does so after intensive research and after its toxicity has been confirmed.
- b. Identifying known or potentially hazardous insecticides and developing safety standards for them and methods of control. A list of such harmful insecticides to be annexed as a schedule.
- c. Banning all those insecticides known for their carcinogenicity.
- d. The documents relating to the granting and approval of registration certificates and licences be made public documents. So also the inspector's report and the analysts' report.
- e. The right of a private citizen to set the legal machinery in motion.

2. The Factories Act, 1948 lays down a number of legal obligations on management to ensure that contamination of the work environment is kept to a minimum. Section 13 states that adequate ventilation must be secured and maintained by circulation of fresh air. Section 14 provides that exhaust appliances should be provided as near as possible to the point of origin of the dust or fumes. Section 15 empowers the state government to prescribe standards of humidification. Section 37 prohibits any person from entering a confined space in which dangerous fumes are likely to be present. Before entering such space it has to be certified as safe by an expert. Suitable breathing apparatus has to be supplied to the worker who has to be trained in the use of all such apparatus. Like the Insecticides Act, the Factories Act provides for the appointment of factory inspectors, in order to oversee the working of the various provisions of the Act. However, the powers of the inspectors under Section 9 of the Act are limited by the proviso which states that no person shall be compelled under this section to answer any question or give any evidence tending to incriminate himself. The state government is empowered to appoint certified surgeons under Section 10 to examine workers. Similarly, safety officers are to be appointed by the company in every factory wherein one thousand or more workers are employed. The inspector is empowered to take samples after informing the occupier or the manager. One portion of the sample will be sent to the government analyst for analysis and report thereon. The other will be given to the occupier or manager and another portion kept with the inspector. Finally, the schedule to the Factories Act lays down the list of "notifiable diseases". If a worker suffers from the diseases so listed, the employer will have to pay to the medical practitioner such fee as may be prescribed. The government appointed medical practitioner shall examine the worker concerned and send his report to the Chief Inspector with full details. The list of notifiable disease is extremely limited and does not cover many diseases. The employer is liable to be prosecuted if he fails to abide by any of the provisions of the Act. The Factory Rules lay down in detail the safety regulations and the duties of inspectors, certified surgeons etc.

#### Critical comments

The Factories Act in relation to the contamination of the work place may be divided into four parts:

- i. Provisions relating to safety regulations
- ii. Duties of inspectors and certified surgeons
- iii. List of diseases notified, and
- iv. Penalty

In respect to all these four parts, the Act is most inadequate especially with regard to exposure due to hazardous substances. The government has laid down the maximum amounts of polluting substances that can be allowed into the working atmosphere and these are called threshold limit values (TLVs). TLVs are the limits of contamination which scientists believe that workers may be repeatedly exposed to for an average working day. Each substance is assessed according to how dangerous it is. Testing the atmosphere to decide whether the limits are being exceeded is a highly specialised task requiring sophisticated instruments. Moreover, most of the pesticides manufactured in India are carcinogens, like benzene, which has been banned in western countries. A slight exposure to arsenic can cause skin or lung cancer. There is no "safe" exposure to a cancer agent. Hence the only remedy to prevent exposure of workers is to ban these pesticides which are known for their carcinogenicity. It is therefore, important to list those substances in the plant which are known or suspected to be hazardous. The Maharashtra government has proposed an amendment to the Act to include the definitions of what a hazardous process is. The list will be included as a Schedule to the Act. Provisions must also be made by employers to monitor air contaminants in the work environment, along with a Union representative. Another effective method to reduce and control exposure is to control the hazard at its source, particularly by local exhaust ventilation. Other source controls include modifying a work process or substituting the material by less hazardous substances. No such preventive measures are laid down either by the Act or by the Rules.

Workers are not informed about the hazardous substances to which they are exposed. The inspector's report

is not open for inspection. The labelling of chemicals to which they are exposed are unclear and in a language which cannot be understood by workers. Enormous powers are granted to the inspectors for the granting and approval of licences. Greater attention needs to be placed on the industrial policy relating to licensing and inspection especially since there are innumerable instances of licences being granted without studying plans and specifications. As a result of rampant corruption among government officers, licences to factories are indiscriminately granted with a total disregard for human safety. Moreover, no detailed safety standards have been evolved in several industries, and this is particularly the case in the chemical industries.

Though the section relating to medical check-ups is intended to provide prompt medical attention, it in fact tends to weed out workers on the basis of their health. The pre-employment medical check-ups acts as a screening device. This is particularly acute especially since the workers have no access to medical reports, so cannot verify the reports themselves since all the doctors are appointed by the government. It is extremely difficult for a doctor to act contrary to the policy of the company. Moreover, most of the doctors are not fully qualified as industrial physicians or occupational medical practitioners. The worker may be exposed to a hazardous substance and yet the doctor will certify the worker as fit. This is because most chemical carcinogens have a long latency period (time between initial exposure and the appearance of cancer). By the time a chemical is confirmed as a carcinogen, large number of workers may have already been exposed.

Some of the important related U.S. laws are:

1. The Toxic Substances Control Act, 1976
2. Federal right to know and access to records
3. Hazardous substances information and training act

#### Laws dealing with Environmental Pollution including Food Contamination

Pollution caused by pesticides in the atmosphere is responsible for a number of diseases. Maharashtra and Karnataka recorded the maximum number of pesticide poisoning cases in the country. Because many people in India do not get enough protein in their diet, toxic poisoning is much more dangerous. The passing of the Insecticide Act was a result of large scale food poisoning. According to the results of a two year study by the Indian Veterinary Research Institute presented at the Indian Science Congress, 1985, DDT and BHC were found to be present in fish, meat and eggs sold in the Indian market at levels ranging from 1.0 to 8.01 ppm against the tolerance limit of 2.5 ppm. The "Handigodu Syndrome" of bone degeneration in Malnad of Karnataka has been shown to be due to consumption of fish and crabs from paddy fields sprayed with pesticides.

Rule 65 of the Prevention of Food Adulteration Rules, 1955 lays down the tolerance limit of insecticides in foods. However, most of the foodstuffs contain insecticides which are far in excess of the tolerance limit laid down.

The enforcement machinery under the Prevention of Food Adulteration Act, 1954 is inadequate to prosecute the producers, cultivators and sellers of contaminated foodstuffs.

Pesticides contaminate not only foodstuffs, but water, air and soil also. When present in amounts above safety levels, they can often result in degenerative changes in human beings and their environment. The pesticide sprayers themselves are not covered by any labour legislation. The Factories Act does not apply to them. If incapacitated, they cannot claim compensation under the Workmen's Compensation Act. Farm workers who are engaged in spraying are not protected with any protective equipment.

The release of toxic gas by UCIL plant at Bhopal and the sulphuric acid, sulphur trioxide and oleum leaks from the Shri Ram Foods and Fertilizers plants in Delhi display the potential threat that the chemical industry poses to the public at large.

Section 37 of the Air (Prevention and Control of Pollution) Act, 1981 lays down the permissible standards of the discharge of air pollutants. Contravention of the standards laid down by the Pollution Control Board in respect of air pollutants from factories can entail criminal liability both for the company as well as for its officials. If death is caused due to the leakage, the company can be prosecuted under Section 304-A of the Indian Penal Code - "causing death by negligence". Under this section the court has to examine whether the act of the accused was a rash and negligent act and whether that act was the proximate cause of the death. The mere fact that an accused contravenes certain rules and regulations does not establish an offence under Section 304-A.

Section 336, 337 and 338 of the Indian Penal Code deal with Acts "endangering life or personal safety of others". Thus companies which emit poisonous substance with utter disregard for the life and safety of others can be prosecuted under these Sections.

Section 278 of the IPC deals with making atmosphere noxious to health. However, punishment under this Section is a fine of Rs. 500.

Chapter XIV of the IPC deals with offences affecting public health, safety, convenience, decency and morals and covers punishment for sale of noxious food or drink.

#### Civil Liabilities under the Law of Torts

In cases where persons have died or are injured due to food poisoning or affected due to exposure to hazardous substances, compensation and damages can be claimed by filing a civil suit. Unliquidated damages are allowed to compensate the injured party. Damages can be claimed for personal injury, compensation for future medical care and punitive or exemplary damages. The company would be strictly liable to the victim if the person died or was injured due to an abnormally hazardous and dangerous activity carried out by the company. Thus, the company may have taken the utmost care to prevent the release of the noxious gas, yet the company will be held liable. A company can also be held for gross negligence if it can be shown that it failed to exercise the degree of care demanded by the circumstances. Similarly, manufacturers of a product owe a duty of care to all persons who could conceivably use the product regardless of priority of contract. The manufacturer is strictly liable in tort when an article he places on the market knowing that it is to be used without inspection for defects, is proved to have a defect that causes injury to human beings.

It is impossible to deal in this paper with the law relating to Torts. This branch of law is yet to be developed in India.

The Directive Principles of State Policy as laid down in the Constitution of India also provide for the protection and improvement of environment. Article 21 provides for the protection of life and personal liberty. The right to life includes the right to a clean and healthy environment.



"Oh, it kills some of them alright. There is just one alarming side effect."

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